

# DISEASES of the CHEST

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## Observations on Tuberculosis in the City of Cordoba and Pampa De Achala, Argentina, and Minneapolis, Minnesota\*

FRANCISCO TORRES, M.D., and AGUSTIN CAEIRO, M.D.  
*Cordoba, Argentina*

J. A. MYERS, M.D., F.C.C.P., and F. E. HARRINGTON, M.D.  
*Minneapolis, Minnesota*

Various groups of children and some adults have been examined with reference to tuberculosis in Argentina, and compared with those examined in the City of Minneapolis, Minnesota. Obviously, the percentage of tuberculin reactors in any part of the world is dependent on the number of contagious cases in man and domestic animals that have existed in homes and communities where the test is administered and other places where the tested persons have been. In 1939, the tuberculosis mortality rate in Argentina was 98.3 per 100,000 population. In the Province of Cordoba it was 130.7 and in the City of Cordoba it was 228.1. In the United States in 1939 the tuberculosis mortality rate was 44 per 100,000; in the State of Minnesota it was 30.2 and in the City of Minneapolis 31.7. Therefore, one would expect considerable difference in the percentage of children who have the first infection type of tuberculosis, as manifested by the tuberculin reaction.

Koch's Old Tuberculin was employed in Argentina. The first, second and third doses consisted of one-tenth cc. of a dilution of 1:1000, one-tenth cc. of a dilution of 1:100, and one-tenth cc. of a dilution of 1:10. All tests were administered and interpreted personally by Dr. Agustin Caeiro and Dr. Francisco E. Torres. The tests of the first two dilutions were read at the end of forty-eight hours. Those of the third dilution at the end of seventy-two hours, but if

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there was any question concerning the reaction another reading was made at the end of ninety-six hours. The tuberculin tests were read according to the criteria of the National Tuberculosis Association of the United States. Old Tuberculin in dilutions of 1:1000 and 1:100 was used to test the Minneapolis group.

In some of the groups tested in Argentina single films were made of the chests of both the non-reactors and reactors, but in others films were made of the chests of only the reactors. In the Minneapolis group films were made of the chests of all reactors and some non-reactors. All films of the Argentina groups were viewed by both the Argentina and Minneapolis authors. Whenever shadows were found on the x-ray film which might be due to tuberculosis, a most careful history was taken of the parents of the children as to any possible known source of exposure. When sputum was present it was examined, but if tubercle bacilli were not recovered or sputum was not present, gastric washings were procured and carefully examined for the organisms. A careful physical examination was also made of these cases.

The individuals examined are divided into the following groups:

Group I consists of children in one of the primary schools (Jose Maria Bedoya) operated by the Province of Cordoba. Those who attend this school are from families with good economic status. A total of 174 children, ranging in age from nine to sixteen years, inclusive, were tested with tuberculin. One hundred and two (58.6 per cent) reacted. X-ray film inspection of the chests revealed no abnormal findings in sixty-nine (67.6 per cent). There was increase in the linear markings, particularly in the bases, thought to be due to such conditions as bronchitis and bronchiectasis, in eight children (7.8 per cent). In no child in this group was there evidence of pleural change, such as diaphragmatic adhesions. Sharply outlined, dense shadows, thought to represent calcium deposits, were seen in the lungs of fifteen (14.7 per cent). The first infection type of tuberculosis was observed in the pneumonic stage in six (5.9 per cent). Four (3.9 per cent) were found to have the chronic reinfection type of pulmonary tuberculosis, Table 1.

Group II consists of children who are selected from various schools because of undernourishment, etc., and are sent to Dining Room Number 4, where the Province of Cordoba provides them with lunch; otherwise, they are regular school children. All who attend this dining room receive a general examination weekly by a school physician. Those to be served in this dining room are selected from the schools of the district by a physician who examines all children in the school every month. The examinations consist of taking height and weight each week, together with a complete checkup of each child. In case of any emergency, the nurses call the physician, who

is available at all times. When the physician recommends that a child be sent to the dining room, he makes a list of the abnormal findings and the state of nutrition. This list is then given to the school nurse, who visits the home and determines the living conditions of the child, with particular reference to diet. She then gives the information to the dietitian, who arranges for the necessary food requirements with reference to protein, carbohydrates and fats, and adequate amounts of vitamins are given.

In this group, ranging from seven to fifteen years of age, inclusive, 467 children were tested with tuberculin and 248 (53.1 per cent) reacted. Of the entire group of 467, 338 had x-ray film inspections of their chests, and 190 of these (56.2 per cent) had no abnormal findings. There was evidence of increased linear markings, particularly in the bases, of forty-six (13.6 per cent). In three (0.9 per cent) there was evidence of pleural change, such as diaphragmatic adhesions. Evidence of calcium deposits was seen in sixty-three (18.6 per cent) and in thirty-six (10.6 per cent) there was evidence of primary foci that had not yet calcified. In this entire group of children, no case of reinfection type of tuberculosis was found, Table 1.

Group III consists of children in School Number 11, which is conducted for girls only and is operated by the City of Cordoba. The children who attend the school are from families with small incomes but no arrangement is made for special nutrition, etc. However, this school provides all of the supplies, such as books and uniforms, since the families of the children, themselves, cannot make such provisions.

In this group, ranging in age from six to thirteen years, inclusive, 136 children were tested with tuberculin, of whom fifty-nine (43.5 per cent) reacted. Ninety-six had x-ray film inspections of their chests. Of these ninety-six children no disease was located in the lungs in eighty-four (87.2 per cent). The linear markings were increased, especially in the bases, in five (5.2 per cent). Evidence of calcium deposits were seen in three (3.1 per cent). In six children (6.2 per cent) there was evidence of primary lesions which had not yet become calcified, Table 1.

Group IV consists of children in the Eugenio Garzon School, which is an open-air school for undernourished children. It is located near the center of the population. The children admitted to this school are selected by a physician from various other schools on the basis of poor general condition often resulting from under-nourishment, and their families are not able to provide an adequate diet. In this school the children are provided with three meals each day. They arrive at the school at 8 o'clock in the morning to attend classes and are permitted to work and play in the park and gardens

TABLE 1  
SUMMARY OF RESULTS OF EXAMINATIONS  
OF CHILDREN IN SCHOOLS OF CORDOBA, ARGENTINA

Classification	Number	Reactors		Non-Reactors		X-ray Films		Negative		Findings		Pleural Changes		Primary Focus in		Reinfection Type		Non-Tuberculosis		Diseases	
		No.	Pct.	No.	Pct.	No.	Pct.	No.	Pct.	No.	Pct.	No.	Pct.	No.	Pct.	No.	Pct.	No.	Pct.	No.	Pct.
Jose Maria Bedoya School	174	102	58.6	72	41.4	102	69	67.6	8	7.8	15	14.7	0	0.0	6	5.9	4	3.9	0	0.0	
Dining Room Number 4	467	248	53.1	219	46.9	338	190	56.2	46	13.6	63	18.6	3	0.9	36	10.6	0	0.0	0	0.0	
School Number 11	136	59	43.5	77	56.5	96	84	87.5	5	5.2	3	3.1	0	0.0	6	6.2	0	0.0	0	0.0	
Eugenio Garzon School	75	51	58.6	24	41.4	75	41	54.7	22	29.3	14	18.6	2	2.6	0	0.0	4	5.2	0	0.0	
Colony of Vacations	365	171	46.8	194	53.2	99	30	30.3	42	42.4	31	31.3	0	0.0	3	3.0	0	0.0	2	2.0	
Pampa de Achala Children	71	9	12.7	62	87.3	31	9	29.0	12	38.7	5	16.1	2	6.4	0	0.0	0	0.0	4	12.9	
Total	1288	640	51.1	648	48.9	741	423	54.4	135	18.6	131	17.6	7	0.9	51	6.9	8	1.7	6	0.8	

adjacent to the school. At 5 o'clock in the afternoon food is served, after which the children return to their homes until 8 o'clock the next morning. A well-trained pediatrician devotes most of his time to this school. He is assisted by a nurse, who is also the dietitian. Seventy-five children, ranging in age from seven to thirteen years, inclusive, were tested with tuberculin and fifty-one (58.6 per cent) reacted. All of these children had x-ray film inspections of their chests. In forty-one (54.7 per cent) no evidence of disease was seen in either lung. There was increase in the linear markings in twenty-two (29.3 per cent); in fact, cough is one of the reasons for which children are sent to this special school. There was evidence of calcification in fourteen (18.6 per cent) and pleural changes were seen in two (2.6 per cent). The reinfection type of pulmonary tuberculosis was found in four (5.2 per cent), Table 1.

Group V consists of children in a summer camp or vacation colony at Pampa de Achala. The Province of Cordoba provides four summer camps for children at various altitudes, ranging from sea level to Pampa de Achala, which is about 8,000 feet above sea level. Approximately two months before school closes in the spring, physicians examine children in the various schools in the Province of Cordoba and select those who are eligible to these summer camps. This selection is based on such factors as undernourishment. All selectees must also be free from contagious diseases. The children are fluoroscoped to make sure that there are no gross lesions in the lungs caused by any contagious disease. Each summer camp is provided with a physician, who is well trained in pediatrics, and the physician must live in the building with the children. Each group of children sent to the camp at Pampa de Achala remains for one month. While in the camp the group considered in this study had x-ray films of their chests. Great difficulty was experienced in transporting x-ray equipment to this location and making the films, since no electric current was available and a dynamo had to be provided to generate electricity for this purpose. For this one occasion it was desirable to have x-ray facilities available since, in addition to examining the children in the camp, it was important to learn something about the tuberculosis situation among the native citizens in Pampa de Achala. These native citizens consist almost exclusively of sheep raisers and most of the families have resided in this area practically all of their lives. There is little communication with persons from other areas of the province; in fact, this is almost limited to those who bring in provisions by truck. However, the native boys of Pampa de Achala must have one or two years of military training which is given in Cordoba, Buenos Aires, or some other large city. Following this, they return and reside in Pampa de Achala. Some of the native girls go to the cities to seek employment as domestics,

since they are not educated for other positions. However, after a few months to a year or two they become dissatisfied with their positions and return to Pampa de Achala, where they usually spend the remainder of their lives.

In this group, ranging in age from seven to thirteen years, 365 children were tested with tuberculin and 171 (46.8 per cent) reacted. Because of the technical difficulties, x-ray films of the chest were made of only ninety-nine. The lungs appeared entirely clear in thirty (30.3 per cent). In forty-two (42.4 per cent) the linear markings were increased. Attention should be called to the fact that Pampa de Achala is considered an excellent place for persons with chronic bronchitis and asthma and, therefore, in selecting children for the camps those with such conditions were almost invariably included. In thirty-one (31.3 per cent) there was evidence of calcium in the lungs and in three (3 per cent) there was evidence of primary lesions which had not become calcified. In two children (2 per cent) non-tuberculous pulmonary disease was found; both were due to echinococcus cyst, Table 1.

Group VI. For comparison with the groups of children examined in Argentina we have used the records of children between the ages of six and fourteen years, inclusive, examined at the Lymanhurst

TABLE 2  
RESULTS OF TUBERCULIN TEST ON FIRST EXAMINATION  
PUBLIC HEALTH CENTER, MINNEAPOLIS, MINNESOTA

Age in Years	Total Number	No Test	Children Considered	Non-Reactors		Questionable Reactors		Reactors	
				No.	Pct.	No.	Pct.	No.	Pct.
6	305	2	303	262	86.5	1	0.3	40	13.2
7	317	4	313	253	80.8	2	0.6	58	18.5
8	350	3	347	283	81.6	3	0.9	61	17.6
9	334	0	334	273	81.7	5	1.5	56	16.8
10	287	1	286	221	77.3	1	0.4	64	22.4
11	334	2	332	242	72.9	1	0.3	89	26.8
12	338	3	335	231	69.0	0	0.0	104	31.0
13	321	2	319	224	70.2	0	0.0	95	29.8
14	313	1	312	208	66.7	5	1.6	99	31.7
Total	2899	18	2881	2197	76.3	18	0.6	666	23.1

Health Center in Minneapolis between May 31, 1936, and November 1, 1941. During this time 2,899 children reported for examination. The tuberculin test was not administered in eighteen. Of the remaining 2,881 there were 2,197 (78.26 per cent) who did not react. In eighteen (0.62 per cent) the reaction was questionable and in the remaining 666 (23.12 per cent) there was a definite reaction to tuberculin, Table 2. Of the 2,181 children who did not react to tuberculin, no x-ray film inspection was made of the chest in 1,723. This is because it had previously been determined that there is practically never found any evidence of tuberculosis in the chests of children who do not react to tuberculin. Of the 458 non-reactors who had x-ray films of the chest, the lungs appeared entirely clear in 441 (96.1 per cent). There was evidence of pleural changes in four (0.87 per cent), non-tuberculous pulmonary disease in four (0.87 per cent), and calcification in nine (1.96 per cent). In no child was there found clinical tuberculosis in any form, Table 3.

Among the 666 children who reacted to tuberculin on first examination, no x-ray films were made of the chest in eighteen. Of the

TABLE 3  
NON-REACTORS TO TUBERCULIN THROUGHOUT PERIOD OF  
OBSERVATION  
RESULTS OF EXAMINATION  
PUBLIC HEALTH CENTER, MINNEAPOLIS, MINNESOTA

Age	Total Number	No. X-ray Considered	Number	X-ray Negative		Pleural Changes		Non- tuberculous Disease		Calcification	
				No.	Pct.	No.	Pct.	No.	Pct.	No.	Pct.
6	261	221	40	37	92.5	1	2.5	1	2.5	1	2.5
7	251	213	38	36	94.7	1	2.6	0	0.0	1	2.6
8	279	229	50	49	98.0	0	0.0	0	0.0	1	2.0
9	271	213	58	55	94.8	0	0.0	2	3.5	1	1.7
10	219	167	52	51	98.1	0	0.0	0	0.0	1	1.9
11	242	180	62	61	98.4	0	0.0	0	0.0	1	1.6
12	229	166	63	62	98.4	0	0.0	0	0.0	1	1.6
13	223	173	50	49	98.0	0	0.0	0	0.0	1	2.0
14	206	161	45	41	91.1	2	4.4	1	2.2	1	2.2
Total	2181	1723	458	441	96.1	4	0.9	4	0.9	9	2.0

TABLE 4  
TUBERCULIN REACTORS ON FIRST EXAMINATION  
RESULTS OF EXAMINATION  
PUBLIC HEALTH CENTER, MINNEAPOLIS, MINNESOTA

remaining 648, the lungs appeared entirely clear in 474 (73.1 per cent). There was evidence of pleural changes in eight (1.2 per cent), non-tuberculous pulmonary disease in two (0.3 per cent), and the pulmonary disease was not diagnosed in one (0.2 per cent) because of insufficient examination. Calcium deposits were detected in the lungs, the hilum region, or both, in 137 (21.1 per cent). The first infection type of tuberculosis was seen in the pneumonic stage in fifteen (2.3 per cent). In one child (0.2 per cent) the reinfection type of pulmonary tuberculosis was present on first examination. Seven (1.1 per cent), who had no evidence of clinical disease on first examination, subsequently developed the reinfection type of pulmonary tuberculosis. In five (0.8 per cent) of the 648 children, extrathoracic forms of clinical tuberculosis were present on first examination or developed subsequently, Table 4.

Group VII. The tuberculosis survey in Pampa de Achala is unique in that the natives of this area have little contact with large centers of population; indeed, this appears to be the first time that such a survey has been made in South America among an isolated group, at such an altitude. Therefore, it was extended to older ages than those included in the other groups.

In Table 5 we have shown the incidence of tuberculin reactors

TABLE 5  
RESULTS OF TUBERCULIN TEST IN PAMPA DE ACHALA,  
CORDOBA, ARGENTINA

Age in Years	Total Number	Reactors		Non-Reactors	
		No.	Pct.	No.	Pct.
1 to 5	11	1	9.1	10	90.9
6 to 15	71	9	12.7	62	87.3
16 to 25	32	4	12.2	28	87.8
26 to 65	69	45	65.2	24	34.8
Total	183	59	32.2	124	67.8

RADIOLOGICAL FINDINGS, PAMPA DE ACHALA, CORDOBA,  
ARGENTINA

Tuberculin Test	X-ray Films	Number Considered	Negative		Increased Linear Markings		
			No.	Pct.	No.	Pct.	
183	116	116	43	37.1	46	39.6	
Calcification		Pleural Changes	Primary Foci in Pneumonic Stage		Non-tuberculous Disease		
No.	Pct.	No.	Pct.	No.	Pct.	No.	Pct.
26	22.4	5	4.3	1	0.8	3	2.6

among persons ranging in age from one to sixty-five years. The incidence of tuberculin reactors was low up to twenty-five years, and from twenty-six to sixty-five years it only reached 65.2 per cent. This probably is because the older persons of this area have had more contact with persons in and from more densely populated areas. It is of interest to note that of the 183 individuals tested in all ages, only fifty-nine (32.2 per cent) reacted to tuberculin. Within approximately fifty miles of Pampa de Achala the Santa Maria Sanatorium is located, at a much lower altitude, and in the vicinity of the sanatorium are large numbers of tuberculous patients who migrate to this location because of its altitude and excellent climatic conditions throughout the year. There may be slight mingling of the natives of Pampa de Achala with these patients, as the few who go to the cities pass through this area.

In Table 5 it will be observed that of the 116 persons in Pampa de Achala who had x-ray film inspections of their chests, in forty-three (37.1 per cent) the lungs appeared entirely clear. The linear markings were increased, particularly in the bases, in forty-six (39.6 per cent). Evidence of calcification was seen in twenty-six (22.4 per cent). There was evidence of primary lesions which had not yet calcified in one (0.8 per cent), and pleural changes were seen in five (4.3 per cent). In three individuals (2.6 per cent) echinococcus cyst was found in the lungs. In no case was the reinfection type of pulmonary tuberculosis detected.

Although there has never previously been a study of tuberculosis in Pampa de Achala, it is believed that anyone who develops clinical disease in this area would not survive long or would be forced to leave because of the severe climatic conditions.

#### SUMMARY

The tuberculin test was administered to six groups of children in the Province of Cordoba, Argentina, and to a group of Minneapolis, Minnesota, children. Twelve hundred eighty-eight Argentina children and 2881 Minneapolis children were tested. In the Argentina group Koch's Old Tuberculin was used with the initial dose of 0.1 cc. of a dilution of 1:1000. For those who did not react, a second dose consisted of 0.1 cc. of a dilution of 1:100, and for those who still did not react, a third dose consisting of 0.1 cc. of a dilution of 1:10 was administered. In the Minneapolis group, the same procedure was employed except that the third dose was omitted.

Among the Cordoba children the percentage of reactors to tuberculin ranged from 12.7 to 58.6, the average being 51.1. The low percentage of 12.7 was obtained among seventy-one children ranging from six to fifteen years of age, who resided in Pampa De Achala, where there is little contact with large centers of population. In the

remainder of the Cordoba group the lowest percentage of reactors was 43.5. In the Minneapolis group of 2881 children, 23.1 per cent reacted definitely, and 0.6 per cent questionably to tuberculin.

Among the 1288 Cordoba children, 741 had x-ray film inspection of the chest. Although no attempt was made to determine etiology from x-ray shadows, the films presented evidence of disease which was determined to be the first infection type of tuberculosis in the pneumonic stage in 6.9 per cent, and the reinfection type of pulmonary tuberculosis was diagnosed in 1.7. In 17.6 per cent, small, sharply outlined, dense shadows were seen in the pulmonary parenchyma, the hilum region, or both, which were thought to represent depositions of calcium, bone, or dense fibrous tissue. Among the 648 tuberculin reactors in the Minneapolis group the first infection type of tuberculosis in the pneumonic stage was found in 2.3 per cent. On the first examination the reinfection type of pulmonary tuberculosis was found in 0.2 per cent; however, on subsequent examinations this type of disease appeared in 1.1 per cent. Sharply outlined, small densities were seen in 21.1 per cent.

Apparently the disease has the same characteristics among the children of the two nations, as they tolerate the first infection type of tuberculosis in the same manner and the reinfection type of disease is rare in this age group.

The Pampa De Achala observations are unique. Here is an isolated community of families devoting their lives for the most part to the raising of sheep at an altitude of about eight thousand feet. Of 183 persons tested with tuberculin, the incidence of reactors was only 12.2 per cent among those of sixteen to twenty-five years of age. However, in the older age group, the incidence was 65.2 per cent, while the average for all ages was 32.2 per cent. Of the 116 persons in this group who had x-ray film inspection of their chests there was evidence of first infection type of tuberculosis in the pneumonic stage in 0.8 per cent, but in no individual was there any evidence whatsoever of the reinfection type of tuberculosis. Small, dense, sharply outlined shadows were present in 22.4 per cent.

#### RESUMEN

Se ejecutó la prueba tuberculínica en seis grupos de niños de la Provincia de Córdoba, Argentina, y en un grupo de niños de la ciudad de Minneapolis, Minnesota. Se comprobaron 1288 niños argentinos y 2881 niños de Minneapolis. En el grupo argentino se empleó la tuberculina antigua de Koch en una dosis inicial de 0.1 cc. de una dilución del 1 por 1000. A aquellos que no reaccionaron se les administró una segunda dosis que consistió en 0.1 cc. de una dilución del 1 por 100, y a los que todavía no reaccionaron, una tercera dosis de 0.1 cc. de una dilución del 1 por 10. Se empleó el mismo procedimiento en el grupo de Minneapolis, excepto que se omitió la tercera dosis.

Entre los niños de Córdoba el porcentaje de reactores a la tuberculina fluctuó del 12.7 al 58.6, dando un promedio de 51.1. El bajo porcentaje del 12.7 se obtuvo en setenta y un niños de seis a quince años de edad que vivían en Pampa de Achala, donde hay poco contacto con grandes centros de población. En el resto del grupo de Córdoba el más bajo porcentaje de reactores fue 43.5. En el grupo de 2881 niños de Minneapolis, el 23.1 por ciento reaccionó definitivamente y el 0.6 por ciento reaccionó dudosamente a la tuberculina.

De los 1288 niños de Córdoba, a 741 se les hizo un examen radiográfico del pecho. Aunque no se intentó determinar la etiología tomando en cuenta solamente las sombras radiográficas, las películas presentaron evidencia de enfermedad que se determinó ser tuberculosis de tipo primoinfección en el período neumónico en el 6.9 por ciento, y se diagnosticó tuberculosis pulmonar de tipo reinfección en el 1.7 por ciento. En el 17.6 por ciento se notaron sombras pequeñas, espesas y claramente delineadas en el parénquima pulmonar, la región hilial o ambos lugares, que se juzgó representaban depósitos de calcio, hueso o tejido fibroso espeso. Entre los 648 reactores a la tuberculina del grupo de Minneapolis, se descubrió tuberculosis de tipo primoinfección en el período neumónico en el 2.3 por ciento. En el primer examen se descubrió tuberculosis pulmonar de tipo reinfección en el 0.2 por ciento; sin embargo, en exámenes subsiguientes este tipo de enfermedad apareció en el 1.1 por ciento. En el 21.1 por ciento se observaron pequeñas sombras, claramente delineadas.

Aparentemente, la enfermedad presenta las mismas características en los niños de los dos países, pues toleran la tuberculosis de tipo primoinfección de la misma manera, y el tipo reinfección de la enfermedad es raro en este grupo etario.

Las observaciones llevadas a cabo en Pampa de Achala son singulares. He aquí una aislada comunidad de familias que dedican su vida principalmente a la cría de ovejas en una elevación de unos ocho mil pies. Entre las 183 personas comprobadas con tuberculina, la incidencia de reactores en el período etario de diez y seis a veinte y cinco años fue sólo 12.2 por ciento. Sin embargo, en el grupo de mayor edad la incidencia fue 65.2 por ciento, y el promedio de todas las edades fue 32.2 por ciento. Entre las 116 personas de este grupo a quienes se les hizo exámenes radiográficos del pecho, se descubrió evidencia de tuberculosis de tipo primoinfección en el período neumónico en el 0.8 por ciento; pero en ningún sujeto se encontró evidencia alguna, sea lo que fuere, de tuberculosis de tipo reinfección. En el 22.4 por ciento se observaron sombras pequeñas, espesas y claramente delineadas.

## Cavernous Hemangioma of the Lung: Secondary Polycythemia\*

ALFRED GOLDMAN, M.D., F.C.C.P.  
*St. Louis, Missouri*

Hemangioma of the lung is a rare disease. If the lesion is a large one, the arterio-venous fistula which it in effect produces, may be sufficient to cause a secondary polycytemia. Up to the writing of this paper, there were two case reports of hemangioma of the lung with secondary polycytemia. Recently a third report has been added, and for the first time, a pneumonectomy was done for the tumor, with complete disappearance of the polycytemia and the accompanying symptoms. This report is based on a case of a twenty-two year old male with a cavernous angioma causing a severe secondary polycytemia. A roentgenogram of the chest at the age of five was available showing the lung lesion present at that time. The symptoms, physical signs and course of the disease in all the reports are so similar that a clinical entity of cavernous hemangioma of the lung with secondary polycytemia can be established.

In 1938, Rodes<sup>1</sup> reported the first case of hemangioma of the lung complicated by secondary polycytemia. The patient, a twenty-five year old male, gave a history of cyanosis and dyspnea since childhood with clubbing of the fingers at the age of fifteen. His red blood cell count was 7,540,000, hemoglobin 118 per cent. A roentgenogram of the lungs showed three spherical shadows in both lower lung fields. The patient died of pulmonary hemorrhage and at post-mortem there were found three hemangioma of the lungs, two in the right side, one in the left. The diagnosis was not made antemortem.

Horton and Smith<sup>2</sup> in 1939 reported the second case. Their patient, a forty-seven year old male, gave a history of being a "blue" baby at birth. Clubbing of the fingers and cyanosis were noted at the age of twenty-four. He was first examined by Horton and Smith in 1932 when he showed cyanosis, marked clubbing of the extremities, a red blood cell count of 6,000,000 and hemoglobin of 20.6 grams. He was thought to have polycytemia vera. He was seen again in 1938, when his red blood cell count was 6,740,000, hemoglobin 23.7 grams, blood volume 121 cc. per kilogram. A bruit was heard in the base of his right lung, not heard in 1932. A roentgenogram of the chest showed an infiltration of the right postero-lateral lung field. Injection of a

\*From the Dept. of Internal Medicine, Washington University School of Medicine and Barnes Hospital.

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radiopaque medium in the basilic vein showed this to be a vascular tumor, and a diagnosis of angioma of the lung acting as an arterio-venous fistula was made.

Hepburn and Dauphinee<sup>3</sup> in 1942 reported the third case. This patient was a twenty-three year old female who complained of dizziness, faintness, thickness of speech and dyspnea. Clubbing of the fingers was noted at the age of fifteen. The red blood cell count was 9,600,000; hemoglobin 140 per cent; total blood volume 8500 cc. The arterial blood showed 70 per cent oxygen saturation. A roentgenogram of the chest revealed a shadow in the right middle and lower lobes, but no bruit was heard over this area. A pneumonectomy was done by Drs. Shenstone and Janes. A pathological diagnosis of cavernous angioma was made on the removed lung. The patient made a rapid recovery, the cyanosis and polycythemia disappearing quickly, followed by gradual disappearance of the clubbing and other symptoms.

#### CASE REPORT

E. L., aged twenty-two, entered Barnes Hospital June 19, 1942, and was discharged June 24, 1942. He re-entered the hospital July 28, 1942, and was discharged August 4, 1942.

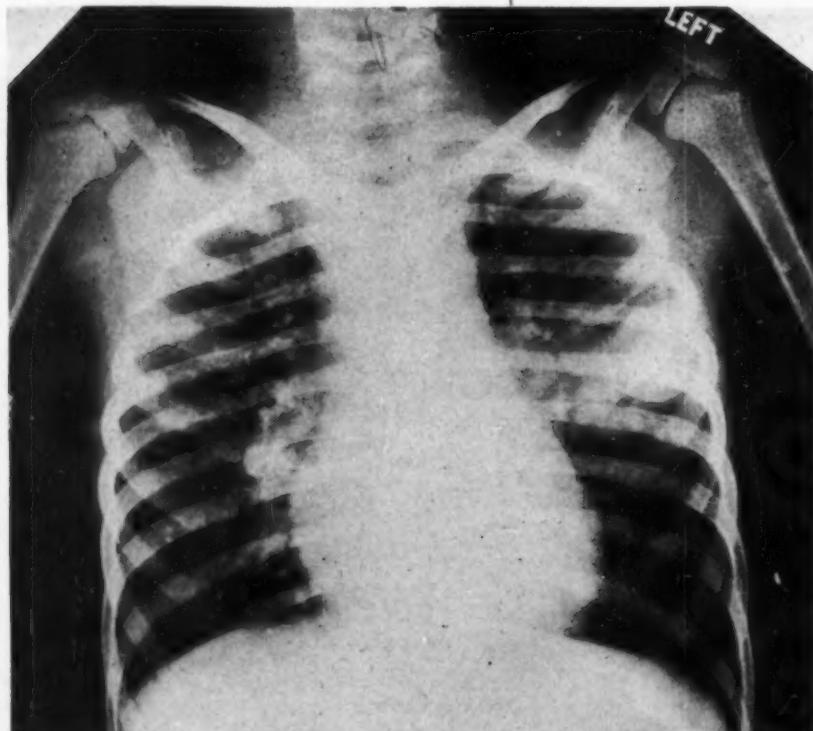


Fig. 1—Bilateral broncho-pneumonia. Note dense shadow in left upper mid-lung.  
(April 5, 1926.)

*Family History*—Mother and father are both living and well. There is no family history of tuberculosis. Patient is an only child.

*Past History*—He had been slightly cyanotic ever since infancy, though apparently in good health up to the age of six. On April 3, 1926, he was admitted to St. Louis Children's Hospital because of a post-measles pneumonia. Cyanosis and clubbing of his fingers and toes were noted then. There was no enlargement of his heart and no murmurs present. His red blood cell count was 6,024,000. A roentgenogram of the lung showed an infiltration in both lungs which was interpreted as bronchopneumonia (Fig. 1). However, a second roentgenogram at the time of discharge showed a clearing of the process in the right lung with a persistent shadow in the left (Fig. 2). He was discharged on April 20, 1926, with the diagnoses of congenital heart disease and bronchopneumonia. He apparently had a fairly normal childhood, and was able to participate in all sports with only slight dyspnea.

*Present Illness*—The patient dates his present illness to 1935, when he started to show marked cyanosis. He developed gradually increasing dyspnea which became severe in 1939. He had frequent frontal headaches. In December, 1940, he had an attack of severe chest pain that lasted two weeks, accompanied by weight loss of twenty pounds. In July and August, 1941, he was treated for polycythemia vera by x-ray and venesecti ons. He gained very little

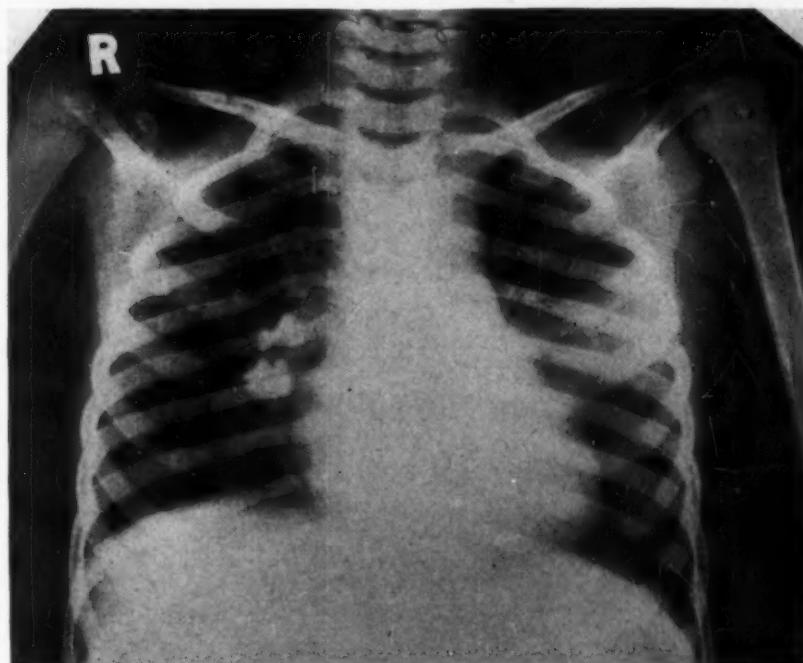


Fig. 2—Clearing of broncho-pneumonia. Shadow in left lung persists. (April 19, 1926.)

relief from therapy. In January, 1942, he had a severe attack of left lower quadrant pain and weakness which invalidated him for nine weeks. His chief complaints on admission were severe frontal headaches, nausea, vomiting, and dyspnea on exertion. There were no episodes of bleeding. He had no cough, sputum, chest pain or recent weight loss.

*Physical Examination*—Temperature 37.4°, pulse 100, respiration 20, blood pressure 110/50. The patient was a tall slender fairly well nourished male with a striking purple-red cyanosis of the lips and nail beds. There were many small purplish macules and papules over his face. There was a small hemangioma on the left upper lid. The conjunctivae were diffusely injected. The heart was not enlarged. There were no murmurs. The lungs were clear on numerous examinations. The spleen was palpable just below the costal margin. There was marked clubbing of the fingers and toes.

*Laboratory Findings*—Red blood cell count 11,450,000; hemoglobin 137 per cent; white blood cell count 4100; platelets 855,000. Blood

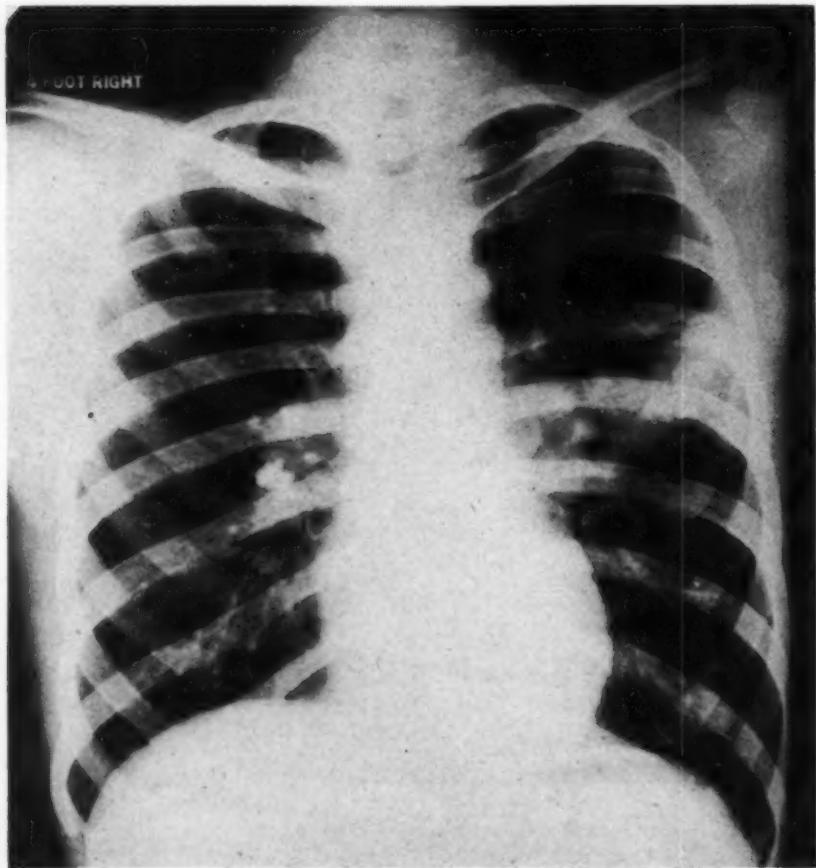


Fig. 3—Dense shadow in left mid-lung field simulating a parenchymal lung lesion.  
(July 29, 1942.)

protein was 5.5, albumen 3.4, globulin 2.1. The Kahn test was negative; urinalysis was normal. A roentgenogram of the chest showed a diffuse infiltration in the left mid-lung field with numerous calcifications in both hilar regions. The x-ray diagnosis was resolving pneumonia (Fig. 3).

*Further Course*—A diagnosis was made of polycythemia vera, congenital. He was discharged on June 24, 1942. On June 26 he received 4.15 millicuries of radioactive phosphorus intravenously which was followed by venous thrombosis at the site of injection. On July 24, 500 cc. of blood were removed by venesection. Because of marked dyspnea, weakness, headaches and pain in both thighs he was readmitted to the hospital on July 28, 1942.

*Second Hospital Admission*—Physical examination showed thrombosis of the left cubital veins; otherwise the physical findings were unchanged from those of the previous entry. The vital capacity was 3200 cc. The circulation time with decholin was 12 seconds, arm to

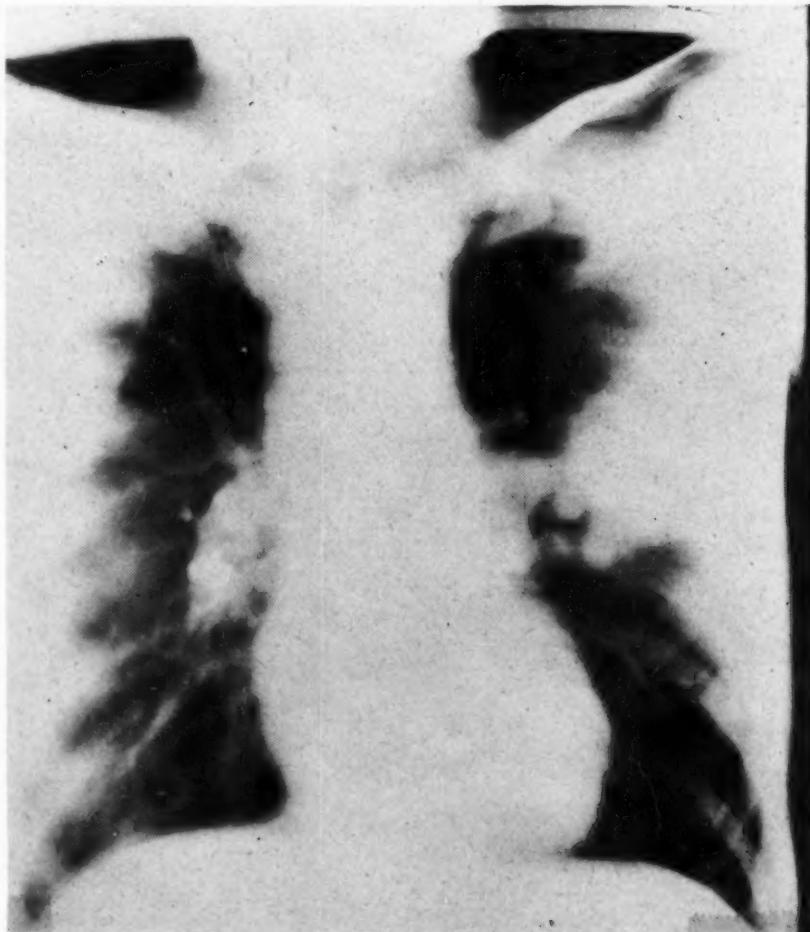


Fig. 4—Antero-posterior laminagram at 6 cms. (July 29, 1942.)

tongue; with ether it was 9 seconds, arm to lung. On July 30, the oxygen saturation of the arterial blood was 70 per cent. Blood volume was 8170 cc.; plasma volume 2450 cc.; cell volume 5720 cc. Second strength P.P.D. tuberculin test was markedly positive. On August 3, the arterial oxygen capacity was 27.4 volume percent, the arterial oxygen content was 16.7 volume per cent, and the venous oxygen content was 9.5 volume per cent. Red blood cell count was 6,850,000, hemoglobin 130 per cent, white blood count 2600. Gastric lavage showed no tubercle bacilli on smear and on guinea pig inoculation. Basal metabolism was plus 21 per cent and plus 24 per cent.

*Roentgenogram Examination*—Laminagrams of the lungs showed a zone of homogenous opacity extending from the fourth to the eighth ribs on the left, best visualized six cms. from the posterior (Fig. 4). In the lateral laminagrams, the opacity was best seen ten cms. from the chest wall (Fig. 5). In the kymogram, there was in the zone of density an intrinsic pulsation which was synchronous with that of the pulmonary artery.

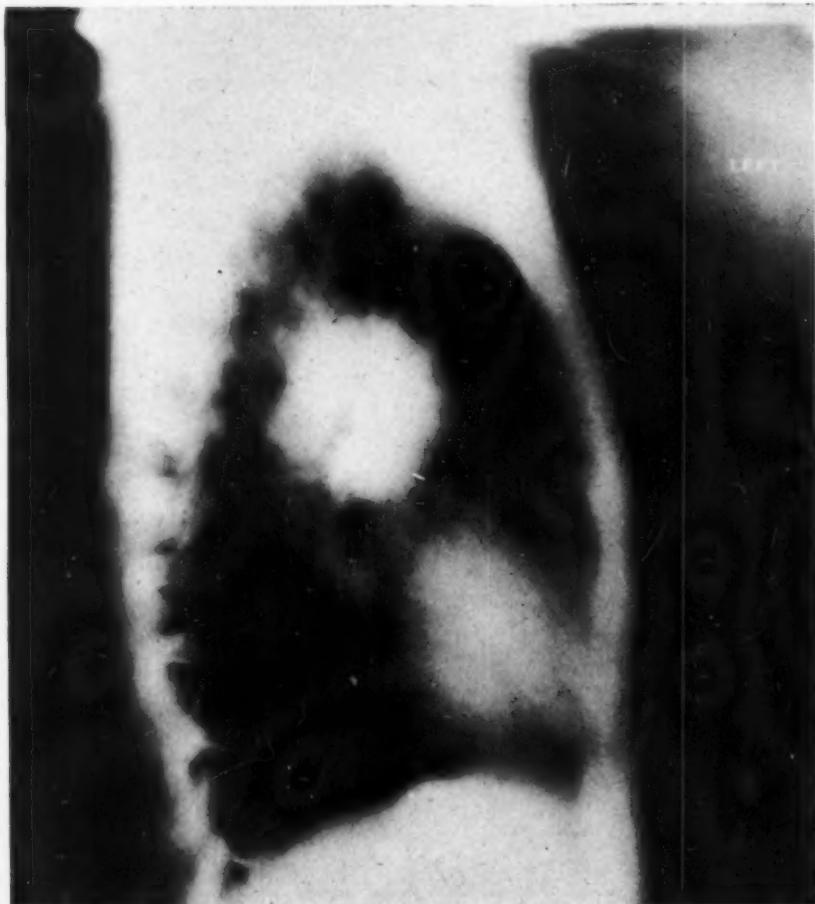


Fig. 5—Lateral laminagram at 10 cms. shows lesion to be a solid tumor-like mass.  
(July 30, 1942.)

*Discussion*.—The case reported is that of a 22 year old male with polycythemia, clubbing of the hands, and a known persistent shadow in the left lung since childhood. The patient had few complaints until the age of 17, when dyspnea and cyanosis became more severe, and by 19, he was more or less incapacitated. There was no evidence of heart disease. The youth of the patient, the clubbing, the severe oxygen unsaturation of the arterial blood and the poor response to the usual therapy for polycythemia vera, rules out the latter diagnosis. The laminagrams show that what appears to be a diffuse infiltrative lung lesion is in reality a solid branching tumor-like mass, which had been present since early childhood. This fact plus the oxygen arterial saturation of 70 per cent suggested the probability that the lung lesion was a cavernous hemangioma of the lung acting as an arterio-venous fistula. The polycythemia, increased blood volume, dizziness, dyspnea, fatigue, and clubbing were all thought to be secondary to the anoxemia. The lack of cardiac hypertrophy was explained by the fact that the lesion involved only the pulmonary circulation. Because of the tendency of the patient to suffer thrombosis, it was considered unwise to inject diodrast. Pneumonectomy was advised, but the patient refused to submit to the operation.

The clinical features of this case and the three previously reported are so similar that it is now possible to describe a clinical syndrome which should suggest the diagnosis of cavernous hemangioma of the lung. The common symptoms are dizziness, headaches, faintness, thickness of speech, fatigue and dyspnea on exertion. Clubbing of the fingers and toes is always present. Polycythemia is constant in the cavernous hemangioma, the degree depending on the size of the arterio-venous fistula. A small angioma of the lung may be insufficient to produce polycythemia.<sup>4,5,6</sup> Oxygen saturation of the arterial blood is low, 70 per cent in our case and Hepburn's and not recorded in the other two. The spleen was palpable only in our case. A striking negative finding is the absence of signs suggesting cardiac disease. The combination of polycythemia, clubbing of the extremities, a low arterial oxygen saturation, negative cardiac findings, plus an unexplained chronic non-progressive pulmonary lesion should lead to the diagnosis of cavernous hemangioma of the lung. While the lesion is a rare one, other cases must have been overlooked in the past. In three of the four reported cases, including ours, the diagnosis was delayed for many years.

#### CONCLUSIONS

- 1) The fourth case of cavernous hemangioma of the lung causing a secondary polycythemia is reported.
- 2) The clinical features of a secondary polycythemia, clubbing of

the extremities, a low arterial oxygen saturation, plus a chronic stationary lung lesion are diagnostic of pulmonary hemangioma.

3) Pneumonectomy or perhaps in some cases lobectomy, is the operation of choice and should result in a clinical cure.

#### CONCLUSIONES

- 1) Se informa sobre el cuarto caso de hemangioma cavernoso del pulmón que causó policitemia secundaria.
- 2) El cuadro clínico de policitemia secundaria, extremidades en forma de maza, baja saturación arterial con oxígeno, más una lesión pulmonar crónica y estacionaria, establece el diagnóstico del hemangioma pulmonar.
- 3) La neumonectomía, o tal vez en algunos casos la lobectomía, es la operación de elección y con ella debe obtenerse la curación.

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## Air Embolism in Artificial Pneumothorax

### Report of 3 Cases

SYDNEY JACOBS, M.D., F.C.C.P.\*  
*New Orleans, Louisiana*

Despite the fact that it does not occur very often, air embolism is still the most terrifying complication witnessed by the physician performing artificial pneumothorax. It is dramatic, unpredictably sudden in onset, and it may be fatal. The number of deaths caused by this unfortunate accident of therapeutic pneumothorax is small, yet the average physician fears them more than he does the infinitely greater number of deaths from pulmonary tuberculosis. In view of this widespread fear, it is reassuring to note that Packard<sup>1</sup> encountered only three instances of air embolism among 100,000 pneumothorax treatments; Matson,<sup>6</sup> four among 20,000 treatments; Tice and Hruby,<sup>7</sup> twenty-eight in an experience of 83,245 refills.

The theory formerly held that some or many of these accidents are due to "pleural shock" is not widely accepted at present. It is much more generally believed that the syndrome is seen only after air has entered a branch of the pulmonary vessels and caused vascular occlusion. This is particularly apt to happen when the pneumothorax needle punctures a fibrotic or caseous lung and establishes a communication between a torn alveolus or bronchiolus and a pulmonary vessel stiffened by disease. As a direct consequence, the patient's symptoms may be purely psychic, chiefly sensory or predominantly motor.<sup>1</sup> At times, there may be a combination of these three types. Common to all is the striking similarity to the clinical picture produced by cerebral vascular occlusion.

Three cases of air embolism, which illustrate very well the types described by Packard, are herein reported. It is worthy of comment that they all occurred within a period of nine days and constituted the only such pneumothorax accidents encountered in the Pneumothorax Clinic of the Flint-Goodridge Hospital where about 2,500 "refills" have been administered to ambulatory negroes since 1936.

*Case 1*—E. J., a 21-year-old female, was first seen 5/30/41 with bilateral fibrocaseous pulmonary tuberculosis. Pneumothorax was begun in the clinic on 6/7/41 because no hospital facilities were available for her. A free pleural space was found without difficulty, and she was given five treatments uneventfully. Partial collapse of the left upper lobe was demonstrated fluoroscopically the day of her sixth treatment, 7/2/41. Manometric fluctuations were free (minus

\*Instructor in Medicine, Tulane University School of Medicine; Junior Consultant, Flint-Goodridge Hospital of Dillard University.  
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9 on inspiration, minus 4 on expiration); after she had received 250 cc. of air, she complained of feeling faint, so the needle was immediately withdrawn and the tip was found to be bloody. There was immediately a generalized convulsion lasting about one minute followed by coma, semi-stupor and cessation of respiration. During this time, oxygen, caffeine-sodio-benzoate and artificial respiration were employed. It is my distinct impression that artificial respiration was the most efficacious of all these measures. After thirty minutes, she recovered consciousness and asked permission to go home. She was persuaded to remain in the hospital overnight where she exhibited no residua and was apparently none the worse for her experience. She stayed at home a few months and then went to the State Charity Hospital where she is now a patient.

The bloody needle tip suggests penetration of the lung, a probable accident where the lung is only partially collapsed. Convulsions with respiratory failure such as this patient had are not usually included as manifestations of "pleural shock" despite Capps' contention that certain instances of cardioinhibitory pleural shock do occur. It is much more likely that this case should be grouped with Packard's class of air embolism with motor symptoms.

*Case 2*—M. O., 32-year-old female with far advanced bilateral pulmonary tuberculosis, was given her initial pneumothorax on the left side 5/30/41. After a few treatments, she was found to have about a 25 per cent collapse of the left upper lobe, but this could not be demonstrated fluoroscopically the day of her sixth treatment, 7/9/41. The pneumothorax needle was inserted, and the manometric fluctuations ranged from minus 5 on inspiration to minus 2 on expiration. She immediately complained of feeling faint; before any air had been administered, the needle was withdrawn and found not to be bloody. The patient was permitted to rest on the treatment table until well enough to arise but she collapsed while walking away. She was picked up and placed on the treatment table complaining bitterly of a headache, but she had recovered completely before any stimulants could be used. She insisted on going home the same morning and had no evidence of injury from this accident when next seen. She refused to return for treatments or to accept a plan for hospitalization. We learned subsequently that she had expired at home some time in January, 1942. Because she had witnessed E. J. in her convulsion, it was at first thought that she was merely, but naturally, unduly apprehensive. In light of the suddenness of onset, perhaps this syndrome ought more logically to be regarded as a milder manifestation of air embolism with purely psychic manifestations.

*Case 3*—J. B., a 22-year-old male, was admitted to the clinic 1/3/41 with bilateral moderately advanced pulmonary tuberculosis. Else-

where a diagnosis of tuberculosis had been made two years previously with one attempt at artificial pneumothorax, but he had not returned for a refill. Right artificial pneumothorax was started uneventfully 1/8/41 and continued in the clinic until 7/11/41 at which time he reported for his twenty-seventh treatment. At fluoroscopic examination that day, complete upper lobe collapse with about 30 per cent collapse of the lower lobe were seen. Free manometric fluctuations were noted, varying from minus 7 cm. on inspiration to minus 4 cm. on expiration, readings almost identical with those obtained for previous treatments. After he had been given about 100 cc. of air, the nurse noted a peculiar expression on his face, so the needle was quickly removed and found not to be blood-tinged. At once there were generalized clonic, succeeded by tonic, convulsions lasting about thirty minutes and followed by complete left hemiplegia. The face was pulled to the right side, the head and chin deviated to the right, and the tongue deviated to the right on protrusion. The knee jerks and Babinski reflex were not elicited, but there was positive ankle clonus with complete loss of motor and sensory function on the left half of body.

Because of cyanosis and respiratory distress, caffeine-sodio-benzoate was given and oxygen administered. Within a few hours his temperature began to rise, reaching 105° by rectum the next day. For about forty-eight hours we despaired of his recovery, but then he began to improve. The fever waned slowly, disappearing by 7/15/41. Concomitantly, sensory and motor functions returned almost exactly as if he were recovering from a cerebral vascular occlusion. A spinal fluid examination was not performed until 7/26/41. At that time the fluid was found not to be under increased pressure, there were only four cells per cubic millimeter with slightly increased protein; no Wassermann test was reported. The blood Wassermann reaction was found to be strongly positive. (He had been given 12 injections of a bismuth salt and 5 of neoarsphenamine for latent syphilis detected at the time of his original examination.) It is of interest that three weeks prior to the above incident and again two weeks prior, he had complained of a "treatment reaction," about which we could learn no details. He was discharged 7/30/41 with only slight paresis of the left upper extremity and even this had disappeared when last seen 1/18/42. He has steadfastly refused to return to the clinic since that date, fearing that antisyphilitic or pneumothorax treatment would be tried once more.

What the "treatment reactions" were or whether they had anything to do with his pneumothorax accident has never been ascertained. Since he had definite evidences of a lesion in the internal capsule (posterior limb), there is little reason to doubt the diagnosis of air embolism.

Since we lack adequate sanatorium facilities, we are obliged to institute pneumothorax in the clinic for patients who have far-advanced disease with characteristically extensive fibrosis and caseation. It is evident that puncture of the lung by the pneumothorax needle (as must occur occasionally during the course of clinic sessions) can very easily tear a portion of the lung parenchyma and permit air to flow into a pulmonary vein. It is really astonishing that the accident does not occur more often than has been reported. Not infrequently, after an unsuccessful attempt at pneumothorax, the tip of the needle will be bloody indicating that trauma has occurred—and yet no complication follows. Obviously, the unknown factor called "chance" must play a large role.

At the same time, it is difficult to explain the occurrence of three instances of air embolism within a period of three days except by "chance." The possibility of procaine sensitivity was considered but rejected because all patients had previously failed to react adversely when procaine was used for skin analgesia. Contamination of the procaine solution was suspected. Dr. Erwin Nelson, professor of pharmacology, Tulane University, kindly had the clinic supply analyzed and found it to be as labelled, a 2 per cent solution of procaine, free of all foreign matter. He informed me that no known toxin can cause a train of symptoms such as reported, immediately on injection. The operative technic employed is the same as that used since the clinic was first established; all the customary precautions, especially those enumerated by Barnwell,<sup>2</sup> are observed. After the first accident, a most careful attention was paid to these safeguards, and yet this did not avert the second and the third instances of air embolism. The temperature, humidity and other environmental circumstances were practically identical with those under which the clinic has been operated the past six years without any such untoward event; therefore, these could not well be implicated.

A year has elapsed since these complications, the clinic has been conducted in its previous manner, and no instance of air embolism or pneumothorax accident has been noted. It is therefore assumed that the three patients cited were simply those unfortunate few who have air embolic phenomena despite all the safeguards which may at present be exercised. That they occurred within a limited number of days is entirely fortuitous and has no relation to any assignable cause.

#### SUMMARY

Three cases of air embolism complicating artificial pneumothorax are reported to illustrate the common variations in the syndrome when fatality does not occur. These are the mild psychic manifestations, the moderately severe sensory-motor symptoms and the

extremely severe motor type with signs markedly similar to those of cerebral lesions. Since these syndromes are due to entrance of air into a pulmonary vein, usually because the pulmonary parenchyma is ruptured by the pneumothorax needle, the use of a meticulous technic is the best safeguard against this complication. Nonetheless, even under the most carefully controlled of circumstances, such accidents will occasionally happen. The number of fatalities accordingly produced is definitely too small to justify any curtailment of a collapse therapy program or to justify any physicians from withholding the benefits of artificial pneumothorax from any patient whose pulmonary tuberculosis may conceivably be controlled by this form of therapy.

#### RESUMEN

Se presentan tres casos de embolia gaseosa, complicaciones del neumotórax artificial, ilustrativos de las variaciones comunes de este síndrome cuando no resulta fatal. Estas variaciones son: las manifestaciones psíquicas leves, los síntomas sensoriomotores medianamente graves, y el tipo motor extremadamente grave con signos decididamente semejantes a los de una lesión cerebral. Como quiera que estos síndromes se deben a la entrada de aire a una vena pulmonar, generalmente debido a la perforación del parénquima pulmonar con la aguja de neumotórax, la mejor manera de evitar esta complicación es el emplear una técnica sumamente cuidadosa. Sin embargo, aún en circunstancias lo más cuidadosamente controladas, estos accidentes suceden de vez en cuando. El número de muertes causadas de esta manera es ciertamente demasiado pequeño para justificar una reducción en la aplicación de la colapsoterapia o para servir de justificación al médico en negarle los beneficios del neumotórax artificial a cualquier tuberculoso pulmonar si es concebible que su enfermedad pueda ser controlada por medio de esta terapéutica.

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## Intrapleural Pneumonolysis

LT. COMDR. JAMES E. DAILEY, M.C., U.S.N.R., F.C.C.P.  
*Corpus Christi, Texas*

That at least fifty per cent of pulmonary tuberculosis cases require some form of collapse treatment is now a generally accepted fact. Fundamentally, collapse may be divided into two types: temporary or reversible, and permanent or irreversible. The best example of an irreversible collapse produced by operation is, of course, thoracoplasty. An example of a reversible collapse, and incidentally the first technique devised for collapsing the lung, is pneumothorax. A reversible collapse is of such a nature that after a period of time when the lung is supposedly healed, it may be abandoned and the lung allowed to re-expand, thus resuming most of its original function again.

The choice of performing either of the two types of collapse is dependent entirely upon the type of disease present when the patient is first presented to the chest specialist. Time is a tremendously important factor. No longer can we consider this disease with apathetic tolerance and an attitude of expectancy with the mistaken belief that "time heals everything." We are confronted now with a disease that demands immediate consideration, and, in the majority of instances, active methods of treatment.

If a cavity is present, something must be done immediately to obliterate it completely in a race for time before either a hemorrhage occurs or the inevitable spread of the disease results in a hopeless condition.

At present, pneumothorax collapse is the first method of choice. This technique is successful in only about half the cases in which it is first attempted. By "successful" is meant that the area of lung tissue immediately surrounding the diseased part of the lung is completely freed and retracted away from the bony chest wall, thus permitting the cavity to collapse upon all sides. The circumstance involved in the unsuccessful case is the partial collapse of the involved lung with an area overlying the diseased portion remaining attached to the chest wall by means of adherence of the two pleural surfaces. Time is the pertinent factor in the production of these adhesions. As time goes on, the inflammation in the lung parenchyma may gradually extend to the surface involving the two pleurae and causing them to stick together. When a pneumothorax

is then attempted, the free pleural surfaces will separate and the lung will retract from the chest wall leaving the adherent portions holding fast. As the adhesions thus formed are always overlying the diseased areas in the lung, the collapse obtained will be unsatisfactory. Therefore, delay in the institution of pneumothorax may allow this situation to occur so that when an attempt is made, either a poor collapse or none at all will result.

In the past there was generally practiced a dangerous custom of attempting to stretch these adhesions or break them by forcing air into the pleural cavity under positive pressures. This often created serious complications. If the adhesion broke off near the lung, a tear in the lung tissue occurred resulting in a tuberculous or mixed infection empyema. If a large vessel was incorporated in the adhesion, a serious hemorrhage could occur. Occasionally the adhesion would be thin and would stretch enough to allow the cavity to close. The time element was again forgotten in waiting for these adhesions to stretch, and often during this period the patient would cough infected material from the still open cavity into an unaffected part of the lung creating another cavity. The surgical procedure, intrapleural pneumonolysis, was designed to handle this type of case. The sole object of this operation is to create a completely effective pneumothorax out of a poor one. This is accomplished by severing the adhesions holding the lung to the chest wall. Local infiltration with procaine is used for anesthesia. A special cannula is introduced between the ribs into the pleural cavity and a visual instrument similar to a cystoscope is passed through this cannula enabling the operator to directly view the contents of the pleural cavity. A cautery is then inserted through a second cannula in another interspace and the adhesions are severed by direct vision.

The adhesions encountered will vary in size and shape and there may be any number of them. Some appear long and thin like fiddle strings. Others are short, thick and cylindrical in shape. Still others may appear as accordian pleated sheets that run in all directions in the chest. Some of these sheet adhesions are thin as fine tissue paper while others may measure from one centimeter to bands as thick as the wrist. The thicker ones usually have lung tissue extending out into them for varying distances to the chest wall. This condition may present a very real danger; for if the cautery cuts through pulmonary tissue a bronchial fistula results that will cause an empyema. Thick adhesions may also contain very large blood vessels, which, if they are not thoroughly coagulated before cutting, may cause severe bleeding. Often bands will be found attached to the aorta, the subclavian artery, or to any of the other structures in the mediastinum, in which case great skill is required in freeing them. To avoid cutting lung tissue, the safest practice is to sever all adhesions

close to the parietal attachment and even with the very thick ones it is sometimes necessary to cut through the parietal pleura and perform a dissection in the endothoracic space. When doing this, one must remember to avoid the intercostal structures.

Thus, it can be said that this operation, if performed by a skilled operator who has had considerable experience with the procedure, and who has perfected his judgment as to when and where to cut and when not to cut, is a very minimal one as regards the discomfort to the patient. However, in the hands of one not so experienced, this operation can present far greater dangers than any other major surgical procedure in the chest.

The question occasionally asked is, how soon after initiating a pneumothorax should one advise a pneumonolysis operation. The answer to that is, simply, as soon as possible. When a pneumothorax is started and adhesions can be seen to interfere with the collapse, and the space is large enough for the operator to manipulate his instruments, there is no reason for delay. The more rapidly this is attended to, the quicker the cavity will close and the patient will be started on the road to recovery. There is considerable danger in delaying a pneumonolysis. Besides the well-known hazards of an open cavity, the longer one waits the thicker the pleura becomes and the more difficulty one then has in cutting the bands.

Often adhesions are found so thick that to sever them would expose the patient to the dangers of a bronchopleural fistula by cutting the lung. In many of these instances, it is possible to sever a part of the adhesion at one sitting, then to return in three or four weeks and sever the remainder. During this period of waiting the constant pull of the elastic lung on what is left of a partly cut band will lengthen it, the lung pulling away from the chest wall and making the second attempt a safer one. It is not uncommon to take three and four stages to finish cutting one of these thick adhesions. In many instances the lung may be completely adherent over a wide area of the chest wall. When this situation exists, improvement of the pneumothorax collapse by pneumonolysis is impossible. In this case there is no point in continuing the ineffective pneumothorax any longer. It should be stopped, the lung allowed to re-expand, and some other form of collapse considered immediately.

#### SUMMARY

- 1) Remember the time factor and begin active pneumothorax treatment immediately upon an individual who has a cavity. Don't wait to see what happens to the case with prolonged bed rest. Too often the realization will be accompanied by disappointment and chagrin.

- 2) In about half the cases a pneumothorax will be complicated by adhesions.
- 3) Don't attempt to stretch adhesions by means of a positive pressure pneumothorax.
- 4) Make an attempt to sever them by intrapleural pneumonolysis—again remembering the importance of time—as soon as possible.
- 5) In the hands of an expert, the unfavorable consequences of the operation are insignificant and the complications rare, but when performed by one with little experience, the dangers are very real.
- 6) If it is impossible to improve the collapse by pneumonolysis, abandon the pneumothorax and perform a thoracoplasty.

#### RESUMEN

- 1) Tenga presente que el tiempo es un factor de suma importancia y comience el neumotórax terapéutico activo inmediatamente en todo caso que tenga caverna. No espere ver lo que sucederá tras el encamamiento prolongado, pues con demasiada frecuencia lo descubrirá con desilusión y disgusto.
- 2) En la mitad de los casos el neumotórax será complicado por adherencias.
- 3) No intente estirar las adherencias mediante el neumotórax de presión positiva.
- 4) Intente seccionarlas por medio de la neumonolisis intrapleural—teniendo presente de nuevo la importancia del tiempo—tan pronto como sea posible.
- 5) En manos de un experto las consecuencias desfavorables de la operación son insignificantes y las complicaciones raras; pero cuando la ejecuta alguien con poca experiencia los riesgos que existen son muy ciertos.
- 6) Si es imposible convertir en eficaz el colapso mediante la neumonolisis, abandone el neumotórax y ejecute una toracoplastia.

## Interpleural Communication in Bronchiectasis

FRANK I. TERRILL, M.D., F.A.C.S., F.C.C.P.  
and KENNETH A. TYLER, M.D.\*

*Deer Lodge, Montana*

Ochsner<sup>1</sup> in 1939 reviewed the literature and reported only nine cases of interpleural communication. Weller, at the University Hospital in Michigan, has not encountered a single case in his extensive pathological experience.

Seven of the cases reported by Ochsner from a review of the literature occurred in the course of pulmonary tuberculosis. One case was observed by LeWold in 1926 where, following a spontaneous pneumothorax on the right side, one week later there was, in addition to an increase in the collapse of the right lung, a collapse of the left lung.

The case reported by Ochsner was one of communication between the pleural cavities observed during left transthoracic diaphragmatic hernia repair. There was an absence of the anterior mediastinum, and undoubtedly this case was congenital in origin. Embryologically it is possible to have a communication between the two pleural sacs, since in the third week of embryonic life there is a common pleural-pericardia-peritoneal celomic cavity.

The case reported here is one of communication occurring in a very marked bronchiectasis with atelectasis of the left lower lobe, which is probably of an acquired nature rather than congenital in origin.

**Case Report**—The patient was a white male, 24 years of age. He entered the hospital in August, 1942, for observation and diagnosis. He complained of having a cough with rather profuse expectoration since he was seven years of age. When seven years old he had a pneumonia and was in bed for three months at that time, although there was no history of an apparent lung abscess or empyema. Blood streaking of the sputum had been present frequently since the onset. The sputum had a foul odor. Both the cough and sputum were increased for the past three months. A weight loss of 20 pounds occurred during the past six months.

Physical examination was negative except over the left lower chest where there was some impaired resonance with bronchophony and increased whispered pectoriloquy. No rales were present. The patient's clinical course was uneventful while in the hospital. The

\*Montana State Tuberculosis Sanitarium.

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temperature range was 97° to 99° F., pulse rate 76 to 86, and respiration rate 18 to 20. The laboratory findings were as follows: Four sputum examinations negative for acid fast bacilli; urine: acid, 1.033, alb. negative, sugar negative, 1-3 pus cells, and negative for mucus, epithelial cells, and acid fast bacilli. Blood Wasserman was negative. The blood hemoglobin was 95 per cent, red cells, 4.94 millions, white cells, 11,800, polymorphonuclears 84 per cent, lymphocytes 14 per cent, monocytes 2 per cent. Vital capacity was 3.55 liters. Sedimentation rate was 5 mm/1 hr.

A chest roentgenogram showed an atelectatic left lower lobe with the characteristic marked displacement of the heart and mediasti-

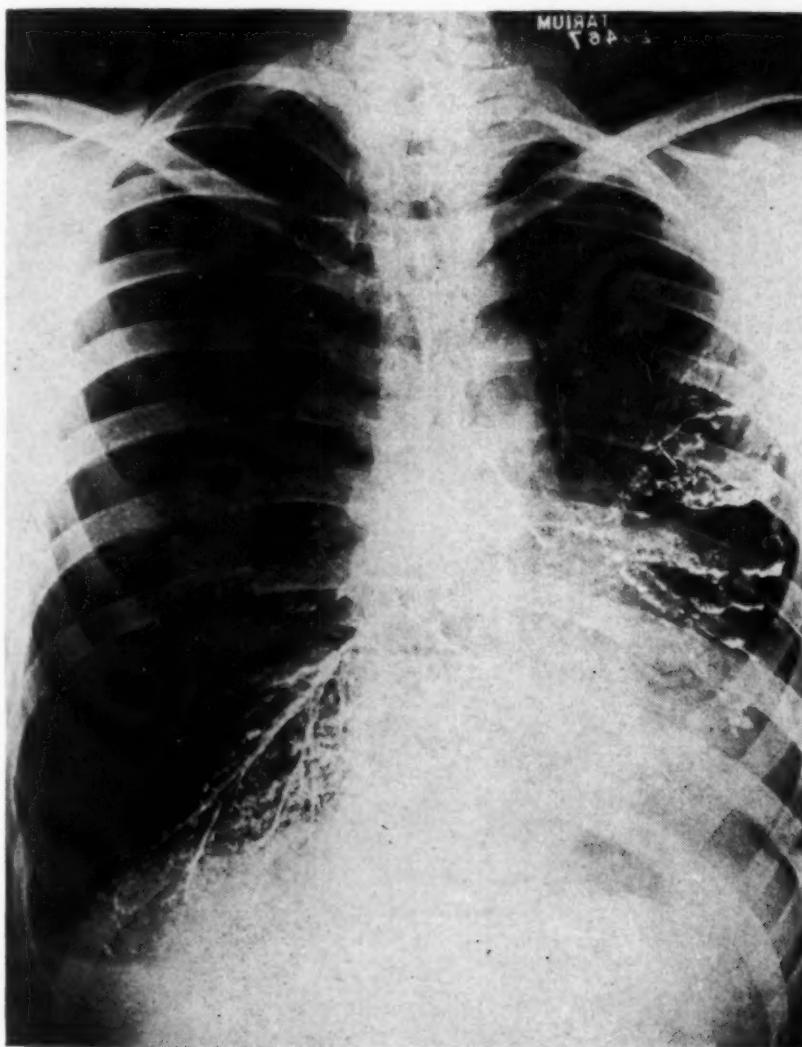


Fig. 1—Bronchogram showing widespread bronchiectasis in the left lung and the displaced lower right lobe to the left without bronchiectasis.

num. A bronchogram, using lipiodol as a contrast medium, showed a marked bronchiectasis present in the atelectatic lower lobe and large dilatations present in the upper lobe. The bronchogram also showed the right lower lobe to be displaced markedly to the left and in close proximity to the atelectatic left lower lobe (Fig. 1).

Because of the widespread and severe bronchiectasis, a left pneumonectomy was advised. In preparation for the procedure a left artificial pneumothorax was begun. After three air instillations some collapse of the left lung was noted, although the pleura was not entirely free. At this time there was also present considerable

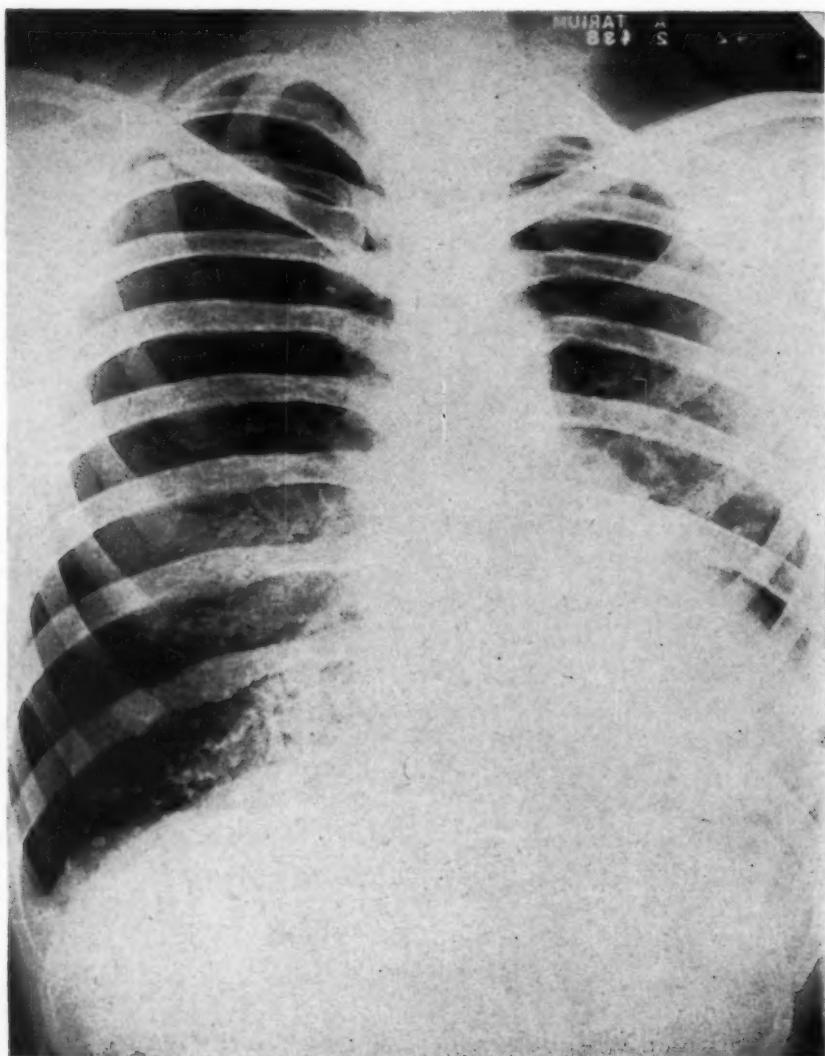


Fig. 2—Antero-posterior roentgenogram of the chest showing partially collapsed left lung with an adherent pleura and also right pneumothorax with a free pleural space, following a left artificial pneumothorax.

pneumothorax collapse of the right lung, with a free pleural space (Fig. 2).

It was concluded that there must have been a communication between the two pleural sacs. It was felt that, while the patient could be kept breathing with positive pressure anesthesia when the left chest was opened, the surgical risk was too great to carry out a pneumonectomy. Besides the anesthetic risk the probability of a bilateral postoperative empyema was a factor in deciding not to subject the patient to this surgical procedure.

#### CONCLUSION

Se llegó a la conclusión de que debe haber existido una comunicación entre los dos espacios pleurales. Aunque hubiera sido posible mantener la función respiratoria por medio de anestesia de presión positiva al abrir el tórax izquierdo, se juzgó que existía un riesgo operatorio demasiado exagerado para llevar a cabo una neumonectomía. Además del riesgo debido a la anestesia, la probabilidad de la aparición de un empiema bilateral postoperatorio fue un factor en la decisión de no someter al paciente a este procedimiento quirúrgico.

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## Contagiousness of Tuberculosis Its Relationship to Compensation Claims

EDGAR MAYER, M.D., F.C.C.P., and  
ISRAEL RAPPAPORT, M.D.  
*New York, New York*

"The doctrine of contagiousness of tuberculosis has now as hitherto its advocates but the general belief of the profession is in its noncommunicability." So did Flint<sup>1</sup> describe the point of view of physicians of the 19th Century.

The oldest dispute in medical history continues in our century and just now it is punctuated by expressions of extremely opposed views.

It is nevertheless true that the pendulum has by now swung in the opposite direction. The prevailing belief of physicians today would best be described by paraphrasing the statement of Flint: Noncommunicability of tuberculosis has now as hitherto its advocates but the general medical belief is in its contagiousness.

*Credo of Noncontagionists:* Noncontagionist advocates are still as blunt as ever. In a recent textbook<sup>2</sup> we read: "Adult pulmonary tuberculosis is not infectious, and is not due to direct deposit in the lung of an exogenous dose of tubercle bacilli." In the British Medical Journal, a lively dispute is now on between contagionists and noncontagionists. The latter<sup>3</sup> deny knowledge of evidence supporting the assumption that adult pulmonary tuberculosis can result by infection from adult to adult.

*Credo of Contagionists:* Among the contagionists are those who with Myers profess even to see no difference between people and cattle as regards contagiousness of tuberculosis. In general it may be said to be characteristic of our time that even the majority of physicians who adhere to the concept of endogenous origin of adult pulmonary tuberculosis believe now in its contagiousness, although these two beliefs are not readily reconciled.

That the two camps can hold such extremely opposing views on basis of equally authentic observations and that current literature abounds in valid arguments for and against contagiousness of adult pulmonary tuberculosis are facts which make the problem more puzzling than ever.

*Tuberculosis can be contagious and noncontagious.* Apparently proper consideration has never been given to the possibility that tuberculosis may be both contagious and noncontagious. Yet when issues are so sharply joined it seems a foregone conclusion that there must be truth on both sides. This is precisely the point we wish to make here.

We will attempt to answer the following questions that naturally arise.

- 1) Is tuberculosis contagious at one and not at another place and time?
- 2) What circumstances make for contagiousness of tuberculosis?
- 3) What are the criteria for adult pulmonary tuberculosis of contact origin?
- 4) What claims for contact tuberculosis are justified under present conditions?

The timeliness and significance of these questions are obvious. The great scientific interest of the first two questions and the medical-legal practical importance of the last two are apparent.

It is evidently time that we physicians reach an understanding upon these questions. Ages ago the laity settled the question in favor of contagiousness of tuberculosis. In courts claims for compensation are granted daily by judges, referees and juries on the lay belief that tuberculosis is always a contagious disease.

To bring order into this, physicians need to set standards for just and equitable decisions in these disputes on the basis of best available knowledge.

*Contagiousness in Relation to Epidemiology:* The changing pathogenesis, pathology and clinical features of tuberculosis with shifting phases of its epidemiology form the basis for its changing contagiousness.

These relationships will be readily understood from the chart included in our recent publication of Journal of American Medical Association. In that chart featuring the upgrade, peak and down-grade epidemiologic phases of tuberculosis the changing character of contagiousness is well illustrated. Lack of space requires us merely to refer to that chart now.<sup>4</sup>

I.) *Is tuberculosis contagious at one and not at another place and time?* Whether tuberculosis is contagious or not is dependent entirely on the epidemiologic conditions prevailing at the time and place.

*Contagiousness in the upgrade phase marked.* Its contagiousness is not even questioned in the first (upgrade) phase of its epidemiology. When the infection appears for the first time in a virgin community, contact and disease follow each other so often and promptly as to leave little doubt about the contagious nature of the infection.

*Noncontagiousness at the epidemiologic peak.* Its contagiousness is first questioned at a time and place, when and where tubercularization has become universal in the community, i.e., in the second phase (peak) of the epidemiology of tuberculosis. In such a fully tubercularized community there is high morbidity and mortality

which has ceased to be related to contact either in time or place. Practically everybody becomes infected already in childhood, but with the exception of infancy these infections do not lead to disease. The prevalent form of tuberculosis now is chronic pulmonary phthisis but this develops long after the infection. As the vast majority of the infected do not develop the disease in spite of continued or repeated exposure it is clear that while the disease of course has its etiologic origin in the initial infection, it is not contact but rather factors inherent in the constitutional characteristics or in the environmental conditions of the individual which play the chief role in its pathogenesis.

In the community at the epidemiologic peak, tuberculosis is infectious but no longer contagious. The infection is so prevalent that most adolescent children and every adult is infected and practically everybody remains exposed to infection more or less continuously. Yet relatively few become diseased. The incidence of disease appears quite independent of the place or time of exposure to known open cases. Such differences of exposure as exist in the community do not explain the spread of the disease. Adult pulmonary tuberculosis is then almost entirely a disease of endogenous origin. The contagiousness of tuberculosis has faded into the background.

*Contagiousness in downgrade phase reappearing.* Recent experience, particularly in this country, has taught us that in the next phase (the downgrade) of its epidemiologic cycle tuberculosis again begins to manifest renewed signs of contagiousness. In this phase of detuberculosis of the community, tuberculosis shows the following striking features. An increasing number of children escape infection altogether or undergo only very mild ephemeral infection leaving evanescent allergy. An increasing number of young adults will come under exposure without having acquired or with already having lost their tuberculin sensitiveness. Of these an increasing proportion will manifest susceptibility to develop disease soon following infection.

There will be no longer interposed a long protracted period of latency so commonly observed before between infection in childhood and chronic phthisis in adult life. Tuberculous disease is again directly referable to recent infection which is mostly traceable to recent contact. All of this makes for recrudescence of contagiousness in this phase of the epidemiology of the disease.

In short, tuberculosis loses its contagious character at the peak but appears as a contagious disease in the first upgrade phase and reappears as such again in the third downgrade phase.

II.) *What circumstances make for contagiousness of tuberculosis?*

**Definition:** Contagiousness is defined as communicability of a disease to another by a person suffering from it. The accepted index of contagion is an expression of the susceptibility and is defined as the proportion of individuals who will if exposed by contact to patient or carrier acquire the disease.

It follows from this definition that the two chief prerequisites for contagiousness of tuberculosis are:

A) A high proportion of individuals susceptible to tuberculosis to the degree that, of those exposed to contact many are liable to develop disease.

B) A high incidence of disease with lesions discharging tubercle bacilli in a manner that those exposed to contact are liable to become infected.

According to the first requirement, tuberculosis will be all the more contagious the greater the proportion of susceptible individuals at the place and time. This is again a matter of the epidemiologic phase.

*Ratio of susceptibles in upgrade phase.* In the first phase we have a virgin community in which practically all who become infected are liable to develop more or less serious disease. The prevalent type of disease is so-called progressive primary tuberculosis which develops in direct relationship with exposure to contact. In such a community the proportion of susceptibles is extremely high.

*Ratio of susceptibles in peak phase.* In the second phase of the epidemiologic cycle, i.e., at the peak of tuberculization, early infection is associated with universal immunization of the community against tuberculosis. Only some children remain susceptible and as infection occurs chiefly in early life these children developing disease usually die out. Those reaching adult age have long overcome their infection but many harbor residues of that old infection which remain capable of exacerbation under the vicissitudes of life. The prevalent type of disease is chronic pulmonary tuberculosis of endogenous origin, the development of which is unrelated to exposure during adult age. The proportion of susceptible adults in the community is very low.

*Ratio of susceptibles in downgrade phase.* In the third phase, i.e., the downgrade of the epidemiologic cycle, the number of tuberculin-negative adults is again rising. The prevalent disease of recently infected adults is chronic pulmonary tuberculosis of exogenous origin which is now again often directly related to exposure during adult age. In short, the proportion of susceptible adults is a considerable factor in the spread of tuberculosis in the community.

That the ratio of susceptibles to resistant in any community or racial group depends solely upon the level of tuberculization has

been demonstrated beyond doubt by repeated observations on the epidemiologic evolution of tuberculosis in freshly exposed peoples. Bushnell gave us a brilliant analysis of previous observations and all recent observations since have borne out his conclusions to the full. In every newly exposed community progressive tuberculization has been associated with marked resistance to tuberculosis in spite of the rising prevalence of infection. Nobody escapes infection at the epidemiologic peak. Mortality and morbidity from tuberculosis is very high but when related to the number infected it is found that in only a small fraction did infection produce disease and this is now of a very latent or chronic type.

How this ratio of susceptibles in the community will be affected by still further detuberculosis remains yet to be determined under the epidemiologic conditions developing now in our midst. To us current observations indicate a trend towards a parallel rise in susceptibility. In recent publications we have endeavored to substantiate our contention that pulmonary tuberculosis is now more often of exogenous origin and in progressive lesions the progression tends to be more prompt as well as more acute than before. This is particularly true for infections in young adults who, with lack of sensitiveness to tuberculin, come under undue exposure.<sup>5</sup>

*Quantitative factors in exposure.* Contagiousness of tuberculosis in the first and third phase is also favored by the character of the lesions which are now often of a type discharging vast numbers of bacilli. This then answers the second of the requirements for contagiousness of tuberculosis above enumerated.

The prevalence of acute forms of rapidly and extensively caseating pulmonary lesions (caseous pneumonias) in the first epidemiologic phase are too well known to require further discussion. In the third downgrade phase a rising tendency of more progressive forms of tuberculosis becomes again manifest particularly in previously uninfected young adults coming under unduly heavy exposure.<sup>4</sup> The marked tendency towards early caseation and rapid liquefaction of acute lesions affords relatively greater opportunities for dissemination of the bacilli in their environment. In addition these lesions contain such enormous quantities of bacilli as to make for more dangerous exposure on contact with such patients.<sup>6</sup> It has been repeatedly demonstrated that rapidly caseating and promptly liquefying lesions contain incomparably greater numbers of bacilli than do fibrocaseous ulcerating lesions, let alone the productive nodular lesions characteristic of chronic pulmonary tuberculosis. That lesions of the former type are more frequent in the first and third phase while lesions of the latter type are peculiar to the second phase of tuberculosis has already been amply explained above. In short then, in the first and third phase of the epidemiologic cycle

heavy dosage favors severe exposure while in the second phase scant dosage favors relatively mild exposure.

III.) *What are the criteria for adult pulmonary tuberculosis of contact origin?*

From his brilliant epidemiologic studies, Bushnell concluded that "epidemics of chronic pulmonary tuberculosis are not possible" and that "primary tuberculosis alone can occur in epidemic form." His implication clearly is that tuberculosis of contact origin is always primary in character. Taking the dictum of Bushnell as a basis, the answer to our third question would seem simply to make primary character of the disease the criterion of tuberculosis of contact origin. In a broad sense it might be said that this is correct. The only truly valid criteria we have for tuberculosis of contact origin are lesions developing promptly following recent conversion from negative to positive tuberculin reaction. This presupposes of course that the individual in question was recently exposed, whether or not the source be known. Accordingly we should be dealing with primary tuberculosis.

On closer analysis, however, the situation is somewhat more complex as the dictum of Bushnell is not quite applicable to the present situation. A generation ago first infections occurred almost exclusively in childhood. The form of primary tuberculosis in adults Bushnell speaks of is that which he observed in the virgin soil of American Indians. Whether progressive or not this form takes the characteristic evolution of childhood tuberculosis. Progression of recent adult infection in form characteristic of adult type tuberculosis was not described by Bushnell, as presumably he had not observed it. The latter mode of progression of a recent presumably first infection is a feature of so-called "adult primary tuberculosis."

*Adult primary tuberculosis in the present epidemiology.* The latter is a newly observed manifestation characteristic of the present downgrade epidemiologic phase of tuberculosis. With regard to this new form we are in an uncertain position. The prevalent opinion is that it represents a primary infection but because its clinical evolution is identical with that of reinfection tuberculosis, we can never be certain of its true character at any time. This is true now even when it is observed following recent conversion of the individuals from negative to positive reaction. In our present environment first infections as well as the allergy they elicit often now become evanescent. Conversion from negative to positive tuberculin reaction no longer is a criterion of a true primary tuberculosis. We are dealing here undoubtedly with an exogenous new infection which is quite primary-like in its origin even if unlike in its evolution.

In recent publications we have emphasized that a special feature

of the present epidemiologic phase of tuberculosis seems to be that chronic pulmonary tuberculosis now may develop promptly from lesions of exogenous origin directly related to recent exposure and contact. The disease which so develops is in every way identical with chronic pulmonary tuberculosis of endogenous origin well known to us from the time when first infections occurred in childhood and progressive pulmonary disease developed in adult age.

The situation is now even more confused by the fact that exogenous and endogenous phthisis occur now alongside of each other and are impossible to tell apart in any case, by any clinical or x-ray features.

We now have the great difficulty of distinguishing between cases of pulmonary tuberculosis which in one instance may represent truly a disease acquired by contact, while in the other instance its development occurred entirely independent of exposure.

The fact is that tuberculosis still is a rather prevalent disease. While our communities are as a whole already far detuberculized they still harbor many "nests" of racial groups or families in which tuberculosis is still somewhere in the transition between the peak and downgrade phases. There are still many cases of endogenous phthisis derived constantly from these nests and from the ranks of the older age groups who had been infected in childhood a generation ago.

It must be concluded that under present conditions the only valid criterion for adult pulmonary tuberculosis of contact origin are progressive pulmonary lesions developing promptly following recent conversion from negative to positive tuberculin reaction.

IV.) *What compensation claims for contact tuberculosis are justified?* Today few claims for compensation for tuberculosis acquired by contact are justified. The trend among physicians involved in these legal proceedings to accept and even to make use of the lay belief that pulmonary tuberculosis is always the result of recent exposure is to be deplored as contrary to best modern medical knowledge.

The fact is that tuberculous infections are still so prevalent that in our more densely populated areas 75 per cent of people have been infected by the age of 30 and nearly 100 per cent are infected by the age of 50 years. Thus the chances of becoming infected with tuberculosis sometime during life are still almost 100 per cent for most of us, regardless of occupation. Exposure and infection lead to disease in but relatively few individuals. These few may be described as "susceptibles." The chances even for these susceptibles to escape infection throughout life are, regardless of their occupation, still rather low even in the environment prevailing now in the greatest part of this country. It should be conceded, however,

that under the present conditions as yet uninfected young people or individuals coming from already highly detuberculized areas are more likely to develop progressive lesions when they become infected in an environment of heavy exposure (hospitals, sanatoria, living places close to open cases of phthisis).

Accordingly claims for compensation for contact tuberculosis may be classified in the following three groups listed in the order of their merit:

1) Professionally exposed hospital personnel who were tuberculin-negative prior to exposure.

2) Young adults (18 to 25) who upon recent exposure developed infiltrative type lesions more or less rapidly progressive, even if knowledge of tuberculin reaction prior to exposure is lacking. Chronic type lesions even in young people suggest old childhood infection unless recent conversion from negative to positive tuberculin reaction proves recent infection.

3) Persons, regardless of age, who recently moved from an environment of known low level of tuberculization, and have suddenly developed lesions of more or less acute character. This includes persons who have recently come from thinly populated sections of the country into new jobs, involving occupational exposure in a heavily tuberculized environment.

Only in the first group of known negative-tuberculin reactions can we say that the claim is justified beyond doubt. The other groups are included for consideration upon the basis of broad interpretation of the evidence to serve justice with utmost fairness. In young adults it may be reasonably assumed that their infection occurred recently because under the present epidemiologic conditions infections rarely take place in childhood. As to the last group it is plausible to assume that in their previous detuberculized environment they may have escaped infection and that their present infection is recent.

Two points, however, must be stressed when considering claims for the last two groups:

1) The contact history must be recent, i.e., the lesions shall have developed within 3 years after the beginning of exposure. All evidence now indicates that in susceptible individuals, lesions develop upon exposure within a relatively short period and that if no lesions develop within 3 years the person is not susceptible and the contact is harmless. Lesions developing later indicate "endogenous phthisis" most likely from childhood infection and unrelated to exposure.

2) All other possible predisposing factors should be ruled out. The history must be thoroughly studied for other factors such as pregnancy, diabetes and other intercurrent disease, trauma, etc., before

claims for contact tuberculosis are to be considered as falling within the last two categories.

#### CONCLUSIONS

Contagiousness is not a definite fixed characteristic of tuberculosis. It varies with the epidemiologic conditions in the environment.

In a highly tuberculized environment tuberculosis is not a contagion.

In a not yet tuberculized or an already detuberculized environment, tuberculosis is contagious.

Our present environment is as a whole sufficiently tuberculized yet so that most cases of pulmonary tuberculosis in our midst are not of direct contact origin.

On the other hand there are already in our midst an increasing proportion of individuals, particularly young people, who develop chronic pulmonary tuberculosis of direct contact origin.

As some of these cases develop in connection with occupational exposure they come up for medico-legal consideration from the standpoint of compensation.

The only truly valid criteria for the latter is a lesion developing upon contact in a person known to have been tuberculin-negative shortly before.

470 Park Avenue  
64 East 58th Street

#### CONCLUSIONES

La contagiosidad no es una característica bien definida e inmutable de la tuberculosis, sino que varía de acuerdo con las condiciones epidemiológicas del ambiente.

En un ambiente tuberculizado en sumo grado la tuberculosis no es contagiosa.

En un ambiente no tuberculizado todavía, o ya destuberculizado, la tuberculosis sí es contagiosa.

Nuestro ambiente actual, tomado en conjunto, se encuentra todavía suficientemente tuberculizado, de modo que la mayor parte de los casos de tuberculosis pulmonar en nuestro medio no deben su origen al contacto directo.

Por otro lado, existe ya en nuestro medio, y va en vías de aumento, una proporción de sujetos, particularmente jóvenes, que contraen tuberculosis pulmonar crónica cuyo origen es el contacto directo.

Como quiera que en algunos de estos casos la enfermedad se desarrolla en sujetos expuestos por razón de su ocupación, estos casos se

presentan a la consideración médico-legal respecto de la indemnización.

El único criterio verdaderamente válido para merecer indemnización es el desarrollo de la lesión consecutiva al contacto en una persona que sabese haber sido tuberculinonegativa poco tiempo antes.

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## Lobectomy for Pulmonary Suppuration—Revealing Foreign Body Retained for Twenty-Six Years (Case Report)

MAJ. LOUIS F. KNOEPP, M.C., F.C.C.P.  
*Fort Benning, Georgia*

In 1895 Paul Claisse found that foreign bodies in the lung frequently result in bronchiectasis. Since that report, many instances of retained pulmonary foreign bodies can be found in the literature, such as: drainage tubing, gauze, sequestra, and aspirated objects. Hedblom removed a piece of wood from a lung abscess which had been present ten years following a buzz-saw accident. Many of these foreign objects are responsible for persistent empyema and chest wall fistulae. The following case is a classical example of both bronchiectasis and broncho-cutaneous fistula from retained rubber tubing.

### CASE REPORT

V. F., a married, white female of thirty-one years, was admitted to the Surgery Service of the Shreveport Charity Hospital, December 9, 1940, because of a persistent operative sinus in the posterior right chest. Her history dated back twenty-six years, at which time she had pneumonia followed by a right empyema. This was drained surgically, but purulent material continued from the site of the wound for some twenty-eight months thereafter.

There was no recurrence of symptoms until the age of thirteen when her sister accidentally struck her old scar, spontaneous purulent drainage occurring therefrom in five days and continuing intermittently until the present time.

She began to menstruate at thirteen and one-half years, with vicarious bleeding from the thoracic sinus. This bleeding has occurred regularly at each period just a few hours before the menstrual flow, and the last eight years has presented premenstrual hemoptysis in addition. She has been pregnant four times during which the bleeding from the sinus and hemoptysis were absent until the termination of pregnancy. During the last five years there has been a gradually progressive productive cough with increasing amounts of mucopurulent sputum.

During the last six weeks hemoptysis and bleeding from the scar has occurred daily. She has also had a decided increase in the amount of sputum, especially in the recumbent position.

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Her past history includes measles, pertussis, mumps, chicken pox, malaria, influenza and pneumonia on two occasions. The first pneumonia resulted in her present complaint; the second occurred in 1937, following which she noted swelling of all her fingers, wrists, and knees. She has two living children, and has had two miscarriages. In 1930 she underwent a pelvic operation for the removal of a tumor which was described as cystic and weighing six and one-half pounds. At this operation her right tube, ovary, and appendix were removed.

*Physical Examination*—Physical examination revealed a slightly obese female, who appeared chronically ill. There was moderate pallor of the skin and mucous membranes. Except for poor oral hygiene, and a well-healed midline abdominal scar, all regions of the body were essentially normal except the thorax and extremities. The blood pressure was 110/70. There was an intercostal scar with a draining sinus over the postero-lateral portion of the tenth rib on the right. Chest expansion was approximately equal, but moist rales were heard over the right lower lung field, both anteriorly and posteriorly. The fingers were all spindle-shaped with moderate limitation of motion. The knees and wrists were moderately swollen but no tenderness was noted.

*Special Tests*—Laboratory findings revealed a moderate anemia, with 3,500,000 red blood cells, 13,450 white blood cells, and a hemoglobin of sixty-three per cent. The Kahn test was negative. Mosenthal renal concentration test revealed a urinary concentration of 1.023 in the first specimen, and 1.020 in the second.

Roentgenograms and bronchograms were performed revealing a bronchiectasis of the right lower lobe, with multiple abscess formation. There was complete obliteration of the costophrenic angle and the lateral half of the diaphragm was not visualized.

Bronchoscopic examination was performed under pontocaine locally on December 10, 1940. The left bronchus was normal in all respects, but on passing the scope into the right stem bronchus, much blood and muco-pus were encountered. In the bronchus intermedius and lower lobe bronchus there was a moderate amount of granulation tissue encircling these divisions. A small amount of purulent material was found in the middle lobe orifice, but it was felt that it had been aspirated from the lower lobe.

*Operation*—A transfusion was given the patient on December 11, 1940, and on December 13, 1940, a right lower lobectomy was performed under intratracheal cyclopropane-oxygen anesthesia. Entrance to the right thorax was accomplished by a posterolateral incision over the seventh right rib, with removal of the rib and incision through its posterior periosteum. There were many dense adhesions between the lower and middle lobes, and between the

lower lobe and chest wall. These were excised, or separated bluntly, and the interlobar fissure between the superior dorsal segment of the lower lobe and upper lobe was found obliterated. The hilus was therefore approached from the accessory fissure, but here, too, there was considerable inflammatory reaction preventing isolation of the vessels. The pulmonary ligament was then incised, and the inferior dorsal vein ligated. Clamps were placed between the superior dorsal segment of the lower lobe and the upper lobe, and the lung tissue transected down to the hilus. The site of the fistula was next approached, and due to the density of the adhesions here, it was transected. A lung tourniquet was placed, and the lobe removed. The stump was sutured with approximately six figure-of-eight braided silk sutures, and the bronchus approximated with individual fine silk ligatures. The fistula site was curetted thoroughly, and two pieces of old rubber drainage tube were seen at the site of the sinus in the lung. All bleeding was controlled, and the thorax was lavaged with saline. Drainage was established through a stab wound and resection of the tenth rib posteriorly, inserting a large-caliber Monk's tubing. The wound was closed in layers with several tension sutures for approximation of the ribs.

*Progress*.—Her immediate postoperative condition was excellent, as fluids and blood had been administered through a cannulated foot vein during the operation. Convalescence was uneventful except for a moderate amount of intercostal bleeding at the tube site on December 19, 1940, for which she was returned to the operating room and ligation accomplished. In addition, a temporary right phrenic crush was performed on December 20, 1940, in anticipation of a prolonged empyema of the residual pleural space. Negative pressure was applied to the drainage tube, and the space diminished to 40 cc. capacity by January 28, when she was discharged from the hospital.

Except for persistence of drainage from the broncho-cutaneous fistula until June, 1940, there was no untoward complication. All sputum became absent after the first week postoperatively, and the swelling of her joints receded by the time she left the hospital. She was seen again in October, 1940, when she stated that she felt excellent, and that the wound had not drained. She believed she was pregnant, but a Friedman test was negative. Examination of her pelvis revealed enlargement of the uterus which was felt to be a fibroid. She refused treatment for this.

*Laboratory Specimen*.—"The specimen is a resection from the pulmonary base measuring 5x6x5½ cm. As nearly as can be determined, it appears that the diaphragmatic surface is represented and there is evidence of adhesions here, as well as on the posterior surface. There is a retraction along one border which continues

with a trough like depression that may represent an obliterated fissure between adjacent lobes. There is a considerable excavation at the lower lateral margin which opens to the exterior and contains a piece of rubber tissue drain. This latter measures in length 5 cm., and 1.5 cm. in its greatest width. The cavity thus formed measures 3.5 cm. in its greatest width, and communicates with a dilated bronchus which has been excised 2 cm. above the main cavity. The lining of the cavity is light red and dark red, granular and nodular. On closer inspection two other dilated bronchi are found to communicate with the cavity. There is rather marked dilatation of the bronchi in the pulmonary tissue surrounding the cavity. As a matter of fact, there is a bronchiectatic cavity measuring 2 cm. in diameter quite close to the line of excision. The intervening pulmonary tissue is very firm, wet, grayish-red and somewhat spongy. There are two other pieces of rubber tubing present in the specimen container that the lung was submitted in. Diagnosis: Bronchiectasis. Chronic interstitial pneumonitis and chronic abscess (cavity containing tube), the lining of which shows squamous metaplasia in places. This last feature appears to be present also in the bronchiectatic cavities not related to the foreign body."

#### SUMMARY

A case report of pulmonary suppuration produced by a retained rubber drainage tube is presented. The tubing had remained in situ for twenty-six years, being found in the lung at operation. Lobectomy with removal of foreign body resulted in cure.

#### RESUMEN

Se presenta un informe relativo a un caso de supuración pulmonar causada por la retención de un tubo de drenaje de caucho. El tubo, que había permanecido in situ por espacio de veinte y seis años, se descubrió en el pulmón durante la operación. Con la lobectomía y extracción del cuerpo extraño se obtuvo la curación.

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## Change in the Rate of Circulation and Venous Pressure Following Collapse Therapy in Pulmonary Tuberculosis\*

OSCAR FEINSILVER, M.D., F.C.C.P.

*Worcester, Massachusetts*

The present work is one of several designed to study some of the fundamental circulatory changes associated with pulmonary collapse. Some of the earlier work relevant to the subject matter here presented is reviewed. Although the dual origin of the blood supply to the lungs is highly significant, as has been demonstrated by Wood and Miller<sup>25</sup> by post-mortem injections of both pulmonary and bronchial arterial circuits, the present study is limited to a consideration of the pulmonary arterial circuit and the alterations produced therein by collapse therapy.

The total output of the right ventricle passes through the lungs en route to the left ventricle. It would appear that when a lung is collapsed, it can no longer carry its entire share of the load to the left heart. Experimentally, it has been shown by Dock and Harrison<sup>5</sup> in 1925 by inducing pneumothorax in rabbits that progressive collapse of one lung was associated with progressive lowering of the quantity of blood flowing through it. For several hours considerable oxygen unsaturation was noted. This condition subsequently disappeared. The determination of the quantity of blood flowing through the collapsed lung was based on the assumption that oxygen was absorbed only in the uncollapsed lung and that the progressive increase in the degree of oxygen saturation of the blood indicated shunting of the blood to the functioning lung. In clinical pneumothorax, likewise, since oxygen unsaturation as a rule does not occur,<sup>17</sup> it is clear that the greater part of the pulmonary arterial blood must be shunted from the collapsed lung to the uncollapsed lung, and from collapsed areas of one lung to uncollapsed areas.

The effect of pneumothorax on the circulation, then, is physiologically comparable to the effect of partial occlusion of the pulmonary artery, since part of the vascular bed is shut off thereby. Parallel changes may, therefore, be expected. Fortunately, the consequences of occlusion of part of the pulmonary arterial circuit have been studied in some detail in experimental animals, and a

\*From the Rutland State Sanatorium, Rutland, Mass., and the Chest Clinic of the Worcester City Hospital, Worcester, Mass.

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great deal, therefore, may be inferred therefrom with respect to the consequences of pulmonary collapse.

What effect does partial occlusion of the pulmonary artery have on the output of the right ventricle?

Working with dogs, Fineberg and Wiggers<sup>6</sup> in 1936 demonstrated that ordinarily reduction of the arterial lumen up to 58 per cent may be brought about without changing the output as manifested by maintenance of a constant systemic blood pressure. In 1932 Gibbon, Hopkinson and Churchill<sup>7</sup> demonstrated that 60 per cent occlusion was the critical level beyond which a fall in blood pressure and a rise in venous pressure occurred. Measurements of the minute volume output were found constant up to the critical level. Ligation of both the right and left main pulmonary arteries was studied by Schlaepfer<sup>22</sup> in 1925, working with dogs and rabbits. Underhill,<sup>24</sup> working with cats in which he ligated the left pulmonary artery, demonstrated the constant maintenance of a normal cardiac output, as well as blood pressure. All of the above workers noted a marked elevation of the pulmonary arterial pressure following occlusion of the pulmonary artery. The latter two, who ligated one of the major branches alone, found the elevation to approximate 40 per cent. Moore, Humphreys and Cochran<sup>19</sup> in 1934 reported inconstant and insignificant changes in the cardiac output following occlusion of either right or left pulmonary arteries in dogs.

Several measurements of the minute volume output of the heart in pneumothorax have been made. These will be referred to for direct evidence. Dock and Harrison<sup>5</sup> showed no appreciable change in rabbits in which pneumothorax was induced. Moore and Cochran<sup>18</sup> in 1933 showed decreases in the cardiac output in animals varying from 21.1 per cent to 50.5 per cent. Their conclusions were based on experiments carried on with six dogs weighing between 22 and 39 pounds, in which quantities varying from 200 to 1150 cc. of air were injected. Determinations were made within two to eleven minutes after completion of the injection. When an initial dose of 1150 cc. is injected into the chest of a dog weighing 28.5 pounds, and determinations made two minutes after completion of the injection show a reduction in cardiac output of 50 per cent, the result is not at all remarkable. Obviously, the dog was not given an opportunity to demonstrate his adaptability to the tremendous initial pneumothorax given him. No clinician would venture any comparable experiment in man, such as administering an initial dose of 5000 cc. of air into an individual weighing 125 pounds.

Hilton<sup>11</sup> in 1933 demonstrated a fall in cardiac output in goats as high as 30 per cent following induction of a moderate pneumothorax. However, he showed at the same time that a small pneumothorax was ordinarily associated in his experiments with a slight

rise in cardiac output. Obviously, here again measurements of cardiac output made promptly after injection of a dose which is too large for the immediate adaptability of the animal yields not unexpected lowered values.

In 1935 Cournard, Bryan and Richards<sup>4</sup> made measurements of the cardiac output of six patients, two of whom had spontaneous, the others, artificial pneumothorax. In only two of these was it claimed that a lowered cardiac output was definitely brought about as a result of the pneumothorax. However, inspection of the figures in these cases reveals the following facts: In one case the value before pneumothorax was 6.7 to 8.7 liters. Since the normal basal value for cardiac output is 3 to 4.6 liters, these figures are twice the normal value. It is not stated whether the patient had fever or not, but it may be assumed that the elevated cardiac output was associated with the elevated metabolism resulting from the disease process. It has been shown by Grollman<sup>9</sup> that the influence of mild malaise and hyperpyrexia on the cardiac output is considerable. It is difficult, therefore, to accept these determinations as representing basal values. After three months of pneumothorax during which time the patient received an 85 per cent collapse of one lung, the value for the cardiac output was found to be 4.0 liters—a normal value. Furthermore, it is to be noted that the intrapleural pressure of this patient was raised in the course of treatment, and maintained on the positive side. A lowered cardiac output is to be expected as a result of this change because of its tourniquet effect on the vena cava which diminishes the return of blood to the heart. The paradoxical movement of the diaphragm noted by the authors is obviously the result of the elevated intrapleural pressure making impossible an adequate differential level between the intra-abdominal pressure and the intrapleural pressure. Thus, the paradoxical movement of the diaphragm of the treated side is associated with the lowered cardiac output but is not responsible for it, as was suggested as a possibility.

The second patient had an initial level of 5.6 liters—again an elevated value—which may be attributed to the disease process. After the first month, during which time he obtained a 50 per cent collapse, the cardiac output fell to 4.2 liters—a normal value. Furthermore, after six months, during which time the collapse was increased from 50 per cent to 90 per cent, the level of the cardiac output was found to be 4.4 liters. Here also a marked rise in intrapleural pressure associated with a rise in venous pressure makes the intrathoracic changes more complex than those resulting from pneumothorax as administered slowly in the treatment of tuberculosis.

Richards, Riley and Hiscock<sup>21</sup> reported observations made in three

cases observed over a period of one, two and five weeks respectively after instituting artificial pneumothorax, demonstrating a decreased cardiac output in this period. None of these patients were at basal conditions. The first had a white blood level of 14,500. No record is made of his temperature. The second two, it is stated, had low fever. Large initial insufflations were given. In one case a reduction in cardiac output from 6.1 to 4.7 liters is noted after the introduction of 900 cc. of air as the initial dose, with the production of tachycardia, increased respiratory rate, and transient cyanosis. The second case shows a reduction of output from an initial level of 4.1 to 2.7 liters. However, there is a subsequent rise to 3.8 in three days, although the degree of collapse is maintained unchanged, as indicated by the constantly low vital capacity. This picture suggests that the intrathoracic changes resulting from the initial administration of a volume of air in excess of the compensatory powers of the heart were responsible for the lowered cardiac output, and not the pneumothorax itself. This likelihood is substantiated by the observation of a progressive lowering of the oxygen saturation of the blood from 96 per cent to 87 per cent during the first eleven days of observation, and that cyanosis at rest, increasing after slight exertion was noted. The third case was observed for four and a half weeks. Here an initial level of 4.7 was lowered to 3.8 to 4.0 liters. However, the intrapleural pressure was found elevated to zero, and there was clinical evidence of increased ventilation and tachycardia. None of the above cases are comparable to pneumothorax as commonly given today, where the lung is collapsed gradually. Here oxygen saturation is not diminished; there is no dyspnea or tachycardia; there is no clinical evidence of a lowered cardiac output. It would seem to us that the only conclusion which can be drawn from the above, granting the reliability of the determinations made in the absence of basal conditions, and in the presence of pulmonary disease, that a transient lowering of cardiac output can be observed in pneumothorax in man as in animals when the rate of administration of pneumothorax exceeds the adaptability of the circulatory system to the altered intrathoracic conditions brought about.

The observations of Stewart and Bailey<sup>23</sup> in four cases of spontaneous pneumothorax appear to be conclusive. However, spontaneous pneumothorax occurs quickly, is large in extent. The lowered cardiac output is due not to the extent of the collapse but to the suddenness with which the heart is confronted with changed intrathoracic dynamics. The observation of a lowered oxygen arterial saturation in the single case wherein such measurements were made bears out this explanation.

Since progressive pneumothorax is not in itself responsible for

progressive lowering of the cardiac output by occlusion of a large part of the pulmonary vascular bed resulting therefrom, it may be concluded that almost the total output of the right ventricle is conveyed through the uncollapsed lung. Furthermore, the transfer of a constant volume of blood through one pulmonary artery instead of two can only be brought about through a marked increase in the rate of flow of blood through the single artery.

Conversely, the transfer of blood through the artery leading to the uncollapsed lung at a rate which is not more rapid than that before collapse was instituted, means a lowered cardiac output.

It is clear from the analogy of pneumothorax with ligation of a pulmonary artery that the immediate effect of pulmonary collapse is a rise in the tension within the pulmonary artery. This constitutes an increased load on the muscle of the right ventricle. It has been demonstrated by the experiments on pneumothorax in animals, carried out by Moore and Cochran,<sup>18</sup> Moore, Humphrey and Cochran<sup>19</sup> and those of Hilton<sup>11</sup> that the right ventricle may not be able to adapt itself to this increased load and provide the body with the necessary volume of blood. It is obvious, furthermore, that in pneumothorax and other collapse therapy ventricular compensation is *sine qua non*.

That this compensatory change occurs when one branch of the pulmonary artery is occluded and the blood thereby diverted to the opposite lung was demonstrated experimentally by Churchill<sup>3</sup> who showed that increased oxygen absorption took place in the functioning lung even when no increase in respiratory minute volume occurred. He postulated therefore the following as essential changes: an increased rate of blood flow, opening of new areas of the capillary bed in the functioning lung, and increased pressure within the pulmonary artery.

Of the changes in the functioning lung postulated by Churchill only the change in circulatory rate can be demonstrated with ease in the human. Objective evidence of the compensatory process at any point in collapse therapy may be desired. From what has been said above it is evident that the demonstration of an increased rate of flow of blood from the right ventricle through the pulmonary arteries and veins to the left ventricle indicates that some degree of compensation has taken place. The present work is devoted to a study of the circulation time from the cubital vein to the pulmonary epithelium. Although circulation time does not give absolute evidence of the circulatory rate, since it indicates only the amount of time necessary for the head of the foreign chemical injected to reach a distant point in the body, nevertheless it has been accepted as the most practicable index of the circulatory rate.

Measurements of the circulation time in pulmonary tuberculosis

have been made by several individuals. McIntosh<sup>16</sup> in 1925, measuring the arm to tongue time with decholin, found that the circulatory rate was temporarily lowered somewhat after thoracoplasty. Kal-treider, Fray and Phillips<sup>15</sup> in 1938 found that the decholin time was prolonged in eleven patients 7 to 127 months after thoracoplasty. However, no measurements are available for comparison before operation. Hurst and Brand<sup>14</sup> in 1937 found that measurements in 153 cases of pulmonary tuberculosis of the arm to tongue time by the saccharine method showed no appreciable difference between the groups as a whole, and 81 cases without collapse. Charr and Riddle<sup>2</sup> in 1937 found the circulation time normal in cases of pulmonary tuberculosis with pneumothorax.

Hitzig<sup>13</sup> in 1935 showed by measurements made on eight patients with extensive disease of one lung, that while the venous pressure was normal, the arm to tongue circulation time by the saccharine method and the arm to lung time by the ether method was lowered, indicating an increased blood velocity resulting from mechanisms compensating for the diminished aerating surface of the lungs.

Thus far, therefore, the few measurements made in pulmonary tuberculosis and in the course of collapse therapy have yielded no uniform or understandable data. With a view toward studying the cases undergoing collapse therapy in greater detail, measurements have been made in 21 cases before and after collapse of the lungs was begun.

#### METHOD OF STUDY

The common methods of measuring circulation time in use today were recently summarized by Baer and Slipakoff,<sup>1</sup> and the established norms presented. The drug used for measurement of the arm to tongue time in this work was calcium gluconate, described by Goldberg<sup>8</sup> in 1936 who established the normal as 10 to 16 seconds with an average of 12.5 seconds. Four and five-tenths to 5 cc. of 10 per cent solution of calcium gluconate was injected as rapidly as possible through a No. 18 needle.\* The time required for injection was usually about one second. The results recorded are the total time from the beginning of the injection to the time of the response of the patient as clocked by a stop watch. The arm to lung time was measured by the ether method described by Hitzig.<sup>12</sup> Ordinarily, two determinations of each circulation time were recorded, one after another. It was shown by Hitzig that the determinations

\*Since the completion of this work the author has revised his technique. Now 2.5 cc. of a 20 per cent solution of calcium gluconate is used instead of the previously used 10 per cent solution. The time required for the injection of this small quantity is negligible if done rapidly through a No. 18 needle. Twenty per cent calcium gluconate (neo-calglucogen) has been kindly supplied to the author for this purpose by the Sandoz Chemical Company.

obtained of either the arm to lung time or arm to tongue time may be slightly modified by breathing, rapid breathing increasing the circulatory rate, and diminished breathing decreasing it. Differences as high as 2.5 seconds may be obtained thereby. In the series presented, the time of the injection was made to coincide with the onset of inspiration. Ordinarily agreement within 0.5 seconds between the two determinations was obtained. Any measurements differing by more than two seconds were discarded as unreliable. All determinations were made in a uniformly warm room between two to four hours after breakfast, after the patient had rested in a recumbent position for at least fifteen minutes. No measurements were recorded in patients running fever other than an occasional 99° F., and none were recorded in patients showing undue emotional reactions. Likewise, no measurements were included in individuals showing an anemia greater than 10 per cent from normal.\*\*

In each case simultaneous measurements of the vital capacity and venous pressure on each side were made. Measurements of the latter were made by the direct method. The distance between the height of a column of saline connected to the No. 18 needle within an antecubital vein, and the level of the right auricle considered as 5 centimeters below the sternum at the fourth intercostal space was taken as the venous pressure. All measurements were checked at least twice by repeated refilling of the manometer with saline from a reservoir connected with it.

The results obtained are summarized in three tables wherein the three groups of 21 patients undergoing collapse therapy are segregated, into pneumothorax, thoracoplasty, and extrapleural pneumothorax.

1) *Arm to Tongue Time.* The average for the 3 groups before collapse is 10.8 seconds. After two to four months of collapse therapy the average is 9.8 seconds, a difference of 1.0 seconds. In the group undergoing pneumothorax the initial level of 11.0 seconds becomes 9.7 after two to three months. However, when the mean of the figures available at four to eight months is compared with the mean of the same group before collapse therapy we find a mean value of 9.7 compared with a value of 11.8 seconds, a reduction of 2.1 seconds indicating that the difference in circulation time noted at two to three months, becomes more obvious during the subsequent months. In the group undergoing thoracoplasty the preoperative average of 10.8 becomes 10.1. In the small group undergoing extrapleural pneumothorax the preoperative level of 10.7 becomes 9.2 in two to four months.

\*\*The following figures are considered as normal: red cells: 5.4 million for males, 4.8 million for females; hemoglobin: 16 gms. for males, 14 gms. for females.

2) *Arm to Lung Time.* The average for all three groups before collapse is 6.7 seconds. The average after collapse of two to four months duration is 5.5 seconds. The difference obtained is 1.2 seconds. In the group undergoing pneumothorax, the initial level of 6.5 becomes 5.6 after two to three months. The mean of the figures of this group available at four to eight months is 5.2 seconds as compared with the mean of the same group before pneumothorax of 7.0 seconds, a difference of 1.8 seconds. Here again the impression is obtained that the difference, though slight at two to three months, becomes more obvious in the subsequent months. In the groups undergoing thoracoplasty the average two to three months after the operation was 5.7 seconds as compared with the preoperative level of 7.2 seconds. In the group undergoing extrapleural pneumothorax the average preoperative level of 6.0 is reduced to 4.9 after two to four months.

3) *Venous Pressure.* No significant difference was noted between the preoperative and postoperative levels of venous pressure. Obviously, the postoperative rise in venous pressure observed by Overholt<sup>20</sup> in 1935 is a temporary phenomenon which disappears by the third month. Of the cases undergoing pneumothorax, most show no significant change. Several, however, show slight rises, and two of the group show marked rises: 18 cm. and 29 cm., respectively. In one of these two cases subsequent determination made eight months after the beginning of pneumothorax revealed that the level had been restored to normal although there was no alteration in the degree of his collapse.

4) *Vital Capacity.* This is uniformly lowered. These measurements were made to serve as a check on the degree of collapse undergone.

#### DISCUSSION OF RESULTS OBTAINED

The above figures were submitted to statistical analysis in the following manner. All the values obtained in the three procedures before collapsed therapy were combined, and compared with the combined values obtained after collapse therapy. In the table of pneumothorax figures the first series of measurements obtained after two to three months of treatment was included in the combined post treatment group in order to provide a fairly uniform time interval for this group. The formula used to evaluate the reliability of the differences noted is the following:

$$PE_{(diff)} = \sqrt{PE^2_{average_1} - PE^2_{average_2}}$$

where  $PE_{(diff)}$  is the probable error of the differences between the means, and  $PE_{average}$  is the probable error of the average of each series. The latter is determined from the following formula:

TABLE I  
PNEUMOTHORAX

Case	Measurements made before collapse		Measurements made 2 to 8 months after collapse	
	Arm to Tongue	Venous Pressure (mm Hg)	Arm to Lung	Venous Pressure (mm Hg)
J. M. L.	9.9	5.8	8.1	8.9
J. J. L. C.	11.1	6.7	4.2	5.0
Ho Ho	9.2	8.0	13.3	13.8
Ch Ha	12.0	7.9	7.7	6.5
Ca He	13.0	8.7	7.6	8.4
He He	12.3	7.3	8.9	7.3
A. J.	12.8	6.4	8.6	4.200
H. K.	13.6	8.4	5.0	3500
A. A. L.	9.8	5.5	5.1	7.8
A. L.	12.4	7.7	10.8	9.5
C. N.	12.4	7.7	10.8	9.5
B. J. P.	10.2	6.8	7.5	3800
R. P.	9.6	4.9	5.0	5.8
F. F. R.	11.3	6.0	7.6	3200
A. S.	9.3	4.5	10.2	9.9
I. I. T.	8.4	5.6	9.2	8.3
L. U.	10.3	5.3	6.1	8.9
Mean	11.0	6.5	8.0	7.9
Extremes	8.4	4.8	13.3	13.8
Extremes	13.6	8.0	11.8	7.0
Mean of values comparable with those of 4 to 8 months	11.8	7.0	13.3	13.8

$$\text{PE}_{\text{average}} = \frac{.67456}{\sqrt{N}}$$

where  $\sigma$  is the standard deviation, and  $N$  is the number of determinations made. The value of the ratio of the differences between the means to the probable errors of the differences determined from the above,  $\frac{D}{\text{PE}(\text{diff})}$ , was then referred to tables\* for conversion into terms of reliability.

The differences between the total arm to tongue times as well as the arm to lung times showed a reliability of 99 per cent. Again, the differences between the means of the values obtained for pneumothorax at four to eight months and the corresponding values before collapse therapy were found to have a reliability of 99 per cent and 100 per cent for the arm to tongue and arm to lung determinations, respectively. These are well above the accepted limits of statistical reliability.

Since the change in circulation time observed takes place in a small part of the total arm to tongue route, namely the pulmonary vascular bed, it is felt that a greater difference than that noted, one to two seconds, cannot be expected in a total arm to tongue time of ten to fifteen seconds. The fact that the differences noted in the arm to tongue measurements are approximately equivalent to the differences between the arm to lung measurements indicates that the increase in circulatory rate takes place in the pulmonary arterial circuit. It is felt that this change constitutes the chief compensatory mechanism by which the right heart maintains an approximately constant output in the face of a diminished vascular bed.

The failure on the part of earlier observers to note this increased blood velocity is undoubtedly due to the fact that their determinations were made too soon after collapse therapy was begun. At the onset of these determinations it was noted that measurements made within the first two months frequently were unchanged or even prolonged. Since these subsequently showed an acceleration in blood velocity, additional determinations earlier than two months after the onset of collapse therapy were discontinued. It appears that readjustment on the part of the right ventricle to its increased load requires from one to three months for adequate performance. The exact time required varies in different individuals, and in any particular case, the question as to whether compensation has begun to manifest itself may be answered by comparative determinations of the arm to lung or arm to tongue times. These determinations

\**Statistics in Psychology and Education*, H. E. Garrett and R. S. Woodworth; Longmans, Green and Co., New York, 1930; p. 135.

lose their significance, however, in the presence of anemia, fever, or toxemia.

The rise in venous pressure noted in several cases undergoing pneumothorax is in harmony with the findings of many others. A recent review of previous measurements has been made by Heise and Steidl.<sup>10</sup> From their own determinations the latter conclude that fluid, pneumothorax and thoracoplasty give rise to rare elevations of venous pressure, and usually such elevations are unilateral.

In the present work, of the fifteen cases of pneumothorax whose measurements are tabulated, seven show a rise of 2 to 23 cms. of water above the basal level. These findings are bilateral. It may be said, therefore, that although a rise in venous pressure is not the rule in pneumothorax, it does occur frequently, at least as a temporary phenomenon. Without other evidence of right heart failure, it is impossible to attribute to this explanation the findings observed.

TABLE II  
THORACOPLASTY

Case	Measurements made 1 to 3 months before collapse					Measurements made 2 to 4 months after collapse				
	Arm to Tongue	Arm to Lung	Venous Pressure Collapsed Side	Venous Pressure Uncollapsed Side	Vital Capacity	Arm to Tongue	Arm to Lung	Venous Pressure Collapsed Side	Venous Pressure Uncollapsed Side	Vital Capacity
E. B.	13.6	7.9	10.1	12.0	3400	10.1	5.5	8.1		2200
O. B.	9.3	5.6	12.1	6.8		9.9	4.9	8.6	9.9	2300
E. Car	9.4	8.4	6.5	5.4		12.9	4.5	3.2	6.4	1900
E. Cl	9.9	4.2	10.3	4.7	1700	8.8	4.1	9.5	7.9	1200
M. F.	7.8	5.9	15.0	9.6	1800	7.0	4.2	14.6	10.0	1200
S. H.	9.1	5.9		5.4		9.5	4.5		8.6	
C. K.	13.3	8.1		7.4	2000	12.1	6.6		10.1	1200
R. K.	13.0	6.8	5.5	10.7	2300	10.9	5.0		9.3	1700
C. M.	8.8	5.7	12.0	11.8		9.0	4.8	10.7	9.2	1800
A. P.	7.6		7.3	12.0		6.6		7.5	10.8	1700
E. P.	9.3	7.6	7.7	8.4	2300	9.2	4.6	12.6		1500
C. S.	11.9	7.9	8.8	11.7	3700	11.2	6.0	9.3	14.1	2800
J. T.	14.9	9.0	1.3	2.3	3700	12.9	10.5	.5	1.3	3500
B. U.	12.7	10.8	4.8	3.0	2600	11.8	8.5	7.8	6.3	
Mean	10.8	7.2	8.5	7.9	2600	10.1	5.7	8.4	8.7	1900
Extremes	7.6	4.2	1.3	2.3	1700	6.6	4.1	.5	1.3	1200
	14.9	10.8	15.0	12.0	3700	12.9	10.5	14.6	14.1	3500

## SUMMARY

Evidence is presented from a study of the circulation time in cases undergoing collapse therapy of three types, pneumothorax, thoracoplasty, and extrapleural pneumothorax, that the rate of blood flow is accelerated after collapse. A comparison of the ether time and the arm to tongue time by the calcium gluconate method suggests that this acceleration takes place in the first part of the circuit before reaching the pulmonary endothelium. Determinations made by earlier observers failed to show this change because they were made too soon after the institution of collapse therapy, before the compensatory forces responsible for the accelerated blood flow had had opportunity to become effective. Venous pressure measurements showed no alteration two to three months after surgery was performed. In the course of pneumothorax a transient rise is frequently observed.

## RESUMEN

Se presenta la evidencia obtenida en un estudio del tiempo de circulación llevado a cabo en casos a los que se les aplicó uno de tres tipos de colapsoterapia, neumotórax, toracoplastia o neumotórax extrapleural. Este estudio demuestra una aceleración en la circulación sanguínea consecutiva al colapso. La comparación del "tiempo del éter" con el "tiempo del brazo a la lengua" obtenido mediante la técnica del gluconato de calcio, indica que la aceleración tiene lugar en la primera parte del circuito antes de que la sangre llegue al

TABLE III  
EXTRAPLEURAL PNEUMOLYSIS

Case	Measurements made 2 to 3 months before surgery					Measurements made 2 to 4 months after surgery				
	Arm to Tongue	Arm to Lung	Venous Pressure Collapsed Side	Venous Pressure Uncollapsed Side	Vital Capacity	Arm to Tongue	Arm to Lung	Venous Pressure Collapsed Side	Venous Pressure Uncollapsed Side	Vital Capacity
M. C.	9.3	4.9	12.8	14.2		8.7	3.8	7.5	7.6	
Bilateral J. F.	11.0	5.7	7.7	7.0	2500	8.1	4.8	11.0	9.0	2000
H. M.	7.5	4.6			2000	7.5	4.2			1400
Bilateral W. M.	15.0	8.6	7.5	9.5	3600	12.5	6.9	7.5	9.5	2900
Mean	10.7	6.0	9.3	10.2	2700	9.2	4.9	8.7	8.7	2100
Extremes	7.5	4.6	7.5	7.0	2000	7.5	3.8	7.5	7.6	1400
	15.0	8.6	12.8	14.2	3600	12.5	6.9	11.0	9.5	2900

endotelio pulmonar. Las determinaciones realizadas anteriormente por otros investigadores no demostraron esta aceleración debido a que se hicieron demasiado pronto subsiguiente a la aplicación de la colapsoterapia, antes de que las fuerzas compensatorias responsables por la aceleración de la circulación sanguínea tuvieran la oportunidad de hacerse sentir. Mediciones de la tensión venosa no mostraron alteración alguna dos o tres meses después de la ejecución de operaciones quirúrgicas; pero se observó a menudo una elevación transitoria en los casos de neumotórax.

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## EDITORIAL

### STATISTICS

In this issue there appears a contribution by Dr. J. A. Myers and Dr. F. E. Harrington, Minneapolis, Minnesota, and Dr. Francisco Torres and Dr. Agustin Caeiro, Cordoba, Argentina, reporting on tuberculosis in the Argentine. It calls to one's mind an article which appeared in *Harpers Magazine* in July, 1942, entitled "How Latin Americans Die," by Charles Morrow Wilson. At the time I read Mr. Wilson's article it seemed a very timely one and thought provoking.

In connection with Dr. Myers' and co-authors' paper, I suggest that the article above referred to in *Harpers Magazine* be reviewed by those interested in the tuberculosis problem in South America. It is well written and interesting, but, in my opinion, displays definite and unjustifiable "alarmist" tendencies. We are reminded that modern transportation and the necessities of the present struggle bring the ills of Latin America to our own front yard. It points out that members of our armed forces, our engineers, scientists and merchants are sent to Latin America in ever-increasing numbers and that each individual will be a menace to the health of his family and friends upon his return.

This is somewhat exaggerated and in part scare-mongering. Unfortunately, it is impossible to fight a global war or any other war in which disease is not one of the enemies encountered by both sides. Diseases such as influenza, meningitis, typhus and some of the insect-born tropical diseases we can accept because we must. But, while the dangers from disease are being emphasized we must not forget some of the optimistic aspects such as the effective control of water-borne diseases and the successful immunization against a host of others.

Tuberculosis, one of the diseases mentioned as having a high incidence, need not be too great a hazard. Unlike other infectious diseases, tuberculosis in the adult results from prolonged as well as intimate association with an open case. The circumstances which would bring about such close contact will surely be rare. The greater danger would be from infectious cases existing within the armed forces and this danger has been minimized by careful pre-induction examinations.

The control of each of the diseases mentioned as having a high incidence and mortality has previously been a problem in the United States. Is it too much to hope that a similarly successful control can be had in Latin America in a shorter period of time than it required to effect control in North America? Effective methods of control have been well established and those methods are daily being made available to our friends.

We have no statistics to refute those frightening figures presented by Mr. Wilson. We are willing to grant that as far as they go, they are undoubtedly accurate and official. We only wish to point out that accurate statistics can at times lie convincingly. The South American population is, in great part, distributed in urban centers or in small isolated villages. Many areas, even in the wealthier and more progressive states, cannot be served by physicians or health agencies because it is physically impossible to penetrate the mountainous jungle isolation; therefore, of what value are statistics from such sources? In the large cities the remarkably strict class distinctions among the native population leaves urban statistics open to as much question as those from rural areas.

The wholesome attitude of Latin American nations toward national health is a most favorable sign. The ever-increasing development of the natural resources of our neighbors to the south will have a beneficial rather than harmful effect. Industrialization resulting in greater national income should bring opportunities for many necessary social adjustments. It should bring the advantages of higher standards of living, more evenly distributed educational facilities and health to greater numbers. The outlook seems cheerful rather than gloomy. There is better than an even chance that our proximity will result in better health and higher standards of living for South Americans rather than an increase in disease incidence for us.

Ralph C. Matson, M.D.

## COLLEGE NEWS

### CHAPTER MEETINGS

#### *Southern Chapter*

As this issue of the journal goes to press, the following program is being presented by the Southern Members of the American College of Chest Physicians, meeting jointly with the Southern Medical Association, at Cincinnati, Ohio, November 17-18, 1943:

*Wednesday, November 17*

12:00 Noon

Luncheon Meeting: Roof Garden. Sponsored by the Ohio State Chapter, American College of Chest Physicians. Dr. D. W. Heusinkveld, F.C.C.P., Cincinnati, Ohio, *presiding*.

"A Study of Rejectees for Thoracic Abnormalities," Dr. William A. Hudson, F.C.C.P., and Dr. David Brachman, F.C.C.P., Detroit, Michigan.

2:00 P. M.

Scientific Session on Diseases of the Chest (Roof Garden Foyer). Dr. M. J. Flipse, F.C.C.P., Miami, Florida, *presiding*.

"Chest Diseases in the Aged," Dr. Arnold S. Anderson, F.C.C.P., St. Petersburg, Florida. Discussants: Dr. C. Lydon Harrell, F.C.C.P., Norfolk, Virginia; Dr. David T. Hyatt, F.C.C.P., Little Rock, Arkansas.

"The Relative Importance of the Anatomic and Physiologic Concept in Tuberculosis," Dr. J. D. Riley, F.C.C.P., State Sanatorium, Arkansas. Discussants: Dr. Sydney Jacobs, F.C.C.P., New Orleans, Louisiana; Dr. Alvis E. Greer, F.C.C.P., Houston, Texas.

"New Growths of the Chest," Lt. Col. Carl Tempel (MC) F.C.C.P., Washington, D. C. Discussants: Dr. H. I. Spector, F.C.C.P., St. Louis, Missouri; Dr. Paul H. Ringer, F.C.C.P., Asheville, North Carolina.

"The Occurrence of Pulmonary Tuberculosis in Supposedly Screened Selectees," Col. Arden Freer (MC), F.C.C.P., Washington, D. C. Discussants: Dr. Roy A. Wolford, F.C.C.P., Washington, D. C.; Dr. Frank B. Stafford, F.C.C.P., Charlottesville, Virginia.

"Tuberculosis as a Navy Problem," Lt. Comdr. D. F. Smiley (MC-V (S) USNR), Washington, D. C. Discussants: Dr. Herman E. Hilleboe, F.C.C.P., Washington, D. C.; Dr. Hillis L. Seay, Huntersville, North Carolina.

6:30 P. M.

Cocktail Party: Roof Garden. Given by the Members of the American College of Chest Physicians in Covington, Kentucky, and Cincinnati, Ohio.

7:00 P. M.

Dinner Meeting: Roof Garden. Sponsored by the Board of Regents and the Board of Governors, American Colelge of Chest Physicians. Dr. J. Winthrop Peabody, F.C.C.P., Washington, D. C., *presiding*.

"The Tuberculosis Problem in Puerto Rico," Dr. J. Rodriguez Pastor, F.C.C.P., San Juan, Puerto Rico.

*Thursday, November 18*

9:30 A. M.

Scientific Session on Diseases of the Chest (Ball Room). Dr. Carl C. Aven, F.C.C.P., Atlanta, Georgia, *presiding*.

"Tuberculosis Among Children and Young Adults," Dr. Chester A. Stewart, New Orleans, Louisiana. Discussants: Dr. Charles P. Cake, F.C.C.P., Washington, D. C.; Dr. James L. Bibb, F.C.C.P., Chattanooga, Tennessee.

"The Indications for Total Pneumonectomy," Dr. Evarts A. Graham, F.A.C.S., St. Louis, Missouri. Discussants: Dr. William Reinhoff, Jr., Baltimore, Maryland; Dr. Richard H. Overholt, F.C.C.P., Brookline, Massachusetts.

"Bronchoscopic Kodachrome Motion Pictures of Tracheal and Bronchial Tuberculosis," Dr. Paul H. Holinger, F.C.C.P., and Dr. Ralph G. Rigby, Chicago, Illinois. Discussants: Dr. Dean B. Cole, F.C.C.P., Richmond, Virginia; Dr. Maurice G. Buckles, F.C.C.P., Louisville, Kentucky.

"Lung Resection in Chronic Pulmonary Diseases," Dr. Richard Davison, F.C.C.P., Chicago, Illinois. Discussants: Dr. James L. Mudd, F.C.C.P., St. Louis, Missouri; Dr. George G. Ornstein, F.C.C.P., New York, N. Y.

12:00 Noon

Luncheon Meeting: Ball Room Foyer. Organization of the Southern Chapter, American College of Chest Physicians. Dr. Benjamin L. Brock, F.C.C.P., Waverly Hills, Kentucky, *presiding*.

1:30 P. M.

Conference: College Chapter officials. Dr. Minas Joannides, F.C.C.P., President, Illinois Chapter, Chicago, Illinois, *presiding*.

The meeting is being conducted under chairmanship of the following Fellows: Dr. Champ H. Holmes, F.C.C.P., Atlanta, Georgia, Chairman, Regent's Committee; Dr. Benjamin L. Brock, F.C.C.P., Waverly Hills, Ky., Chairman, Governor's Committee; Dr. Charles M. Hendricks, F.C.C.P., El Paso, Texas, Chairman, Nominating Committee; Dr. Jay Arthur Myers, F.C.C.P., Minneapolis, Minn., Chairman, Scientific Program Committee; Dr. Minas Joannides, F.C.C.P., Chicago, Illinois, Chairman, Conference of Chapter Officials; Dr. John H. Skavlem, F.C.C.P., Cincinnati, Ohio, Chairman, Arrangements Committee; Dr. D. W. Heusinkveld, F.C.C.P., Cincinnati, Ohio, Chairman, Reception Committee.

A further report of the meeting and the organization of the Southern Chapter of the College will be published in the next issue of the journal.

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*New Jersey Chapter*

The Winter Meeting of the New Jersey Chapter of the College will be held at the Martland Auditorium, City Hospital, Newark, New Jersey, Wednesday, December 8, 1943. The following program will be presented:

1. "The War Program of the Tuberculosis Control Section of the U. S. Public Health Service," David M. Gould, M.D., Medical Officer in Charge of Industrial Programs, Tuberculosis Control Section of the U. S. Public Health Service.

2. "Medical Legal Aspects of Pulmonary Diseases," William H. Cox, B.A., LL.B., Senior Member of the firm of Cox & Walburg.

3. "Lung Pathology with Demonstration of Interesting Cases," Harrison H. Martland, M.D., Professor of Forensic Medicine, N. Y. University, County Physician of Essex County, Pathologist, Newark City Hospital.

For further particulars concerning this meeting communicate with Dr. Irving Willner, F.C.C.P., Secretary, New Jersey Chapter, American College of Chest Physicians, 18 Waverly Avenue, Newark, N. J.

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*Indiana Chapter*

The Fall Meeting of the Indiana Chapter of the College was held at the Columbia Club, Indianapolis, on September 28. A luncheon meeting sponsored by the members of the Anti-Tuberculosis Committees of the state medical society was addressed by Dr. H. E. Hilleboe, F.C.C.P., Washington, D. C. Dr. Hilleboe spoke on "Tuberculosis Control in Industry." The paper was discussed by Dr. A. W. Elsten, Anderson, Indiana, and Dr. Philip H. Becker, F.C.C.P., Crown Point, Indiana. Following this discussion, an x-ray conference was held, sponsored by the Indiana Chapter of the College. In addition to the members of the Indiana Chapter and guests from the state of Indiana, the meeting was attended by Dr. Paul A. Turner, F.C.C.P., Louisville, Kentucky, Regent; Dr. Benjamin L. Brock, F.C.C.P., Waverly Hills, Kentucky, Governor of the College for the state of Kentucky; Dr. T. A. Woodson, F.C.C.P., Louisville, Kentucky; and Mr. Murray Kornfeld, Chicago, Executive Secretary of the College. Dr. James H. Stygall, F.C.C.P., Indianapolis, Regent of the College, presided at the luncheon meeting, and Dr. M. H. Draper, F.C.C.P., presided at the x-ray conference. The meeting was well attended.

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*New York State Chapter*

The Fall Meeting of the New York State Chapter of the College was held at the Hotel Biltmore, New York City, October 15, 1943. The following program was presented:

*Morning Session*

"Experience With Pulmonary Resections in Pulmonary Tuberculosis," Dr. Richard H. Overholt, F.C.C.P., Brookline, Massachusetts. Discussion: Dr. J. Maxwell Chamberlain, Ray Brook, New York.

"Idiopathic Spontaneous Pneumothorax, Incidence and Pathogenesis," Dr. George G. Ornstein, F.C.C.P., New York City. Discussion: Dr. Edward P. Egglee, F.C.C.P., New York City; Dr. Moses J. Stone, F.C.C.P., Boston, Massachusetts.

*Luncheon*

"The American College of Chest Physicians as It Stands Today," Dr. J. Winthrop Peabody, F.C.C.P., Washington, D. C., President, American College of Chest Physicians.

*Afternoon Session*

"Rehabilitation of Military Personnel as Carried Out in the Army," Major General S. U. Marietta, F.C.C.P., Washington, D. C.

"A Simple Broncho-Pulmonary Nomenclature and Its Clinical Appli-

cation," Dr. Chevalier L. Jackson, F.C.C.P., Philadelphia, Pennsylvania. Discussion: Dr. Arthur Q. Penta, F.C.C.P., Schenectady, New York.

At a business meeting of the New York State Chapter, following the scientific program, the following resolutions were introduced by Dr. Nelson W. Strohm, F.C.C.P., Buffalo, Governor of the College for New York State:

1) That the following committees be appointed by the President of the Chapter:

- (a) Membership Committee
- (b) Public Relations Committee
- (c) Program Committee
- (d) Nominating Committee

2) That the Membership Dues for the Chapter be advanced from \$2.00 to \$5.00 per year and that the members be given an opportunity to vote on this increase in the annual dues. Final action on this increase to be taken at the next annual meeting of the New York State Chapter to be held in New York City in May, 1944. Both of these resolutions were approved.

One hundred and seventeen physicians registered for this meeting.

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#### *Pennsylvania Chapter*

The Pennsylvania Chapter held its annual meeting at the Bellevue-Stratford Hotel, Philadelphia, on October 6. An x-ray conference was held and the following physicians presented films for discussion: Drs. R. S. Anderson, F.C.C.P., Erie; John S. Packard, F.C.C.P., Allenwood, and Henry A. Gorman, F.C.C.P., Hamburg. The following officers were elected for the ensuing year: Dr. John S. Packard, F.C.C.P., Allenwood, President; Dr. Ross K. Childerhose, F.C.C.P., Harrisburg, Vice-President, and Dr. Edward Lebovitz, F.C.C.P., Pittsburgh, re-elected Secretary-Treasurer.

Dr. Royal H. McCutcheon, F.C.C.P., Bethlehem, President of the Chapter, presided. Dr. Edward Lebovitz, F.C.C.P., Secretary-Treasurer, read the minutes of the last annual meeting. They were unanimously approved. Mr. Murray Kornfeld, Chicago, Executive Secretary of the American College of Chest Physicians, gave a brief talk on College progress. Comments concerning the good and welfare of the Chapter were made by Dr. Frank Walton Burge, F.C.C.P., Philadelphia, former Chairman of the Board of Regents of the College; Dr. C. Howard Marcy, F.C.C.P., Pittsburgh, Regent for the District, and by Dr. John H. Bisbing, F.C.C.P., Reading, Governor of the College for the State of Pennsylvania.

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#### *Michigan Chapter*

The Michigan Chapter of the College held its Fall Meeting at the Statler Hotel, Detroit, Michigan, September 23, 1943. The following program was presented:

1. "Chest Problems in Selective Service," Col. Clarence I. Owen (MCR), Detroit, Michigan.

2. "A Study of Rejectees for Thoracic Abnormalities," Dr. William A. Hudson, F.C.C.P., and Dr. David S. Brachman, F.C.C.P., Detroit, Michigan. Discussant: Dr. George A. Sherman, Lansing, Michigan.

3. "Army Cardiac Examination," Dr. Janney F. Smith, Detroit, Michigan. Discussant: Dr. Saul Rosenzweig, Detroit, Michigan.

Dr. Willard B. Howes, F.C.C.P., President of the Michigan Chapter, American College of Chest Physicians, presided.

*Mexican Chapter*

The Mexican Chapter of the American College of Chest Physicians was organized at Mexico City on September 9, 1943, with 23 charter members. Dr. Edgar Mayer, F.C.C.P., New York City, represented the College at the meeting. During the meeting, the by-laws for College chapters were read and approved and the following officers were elected: President, Dr. Donato G. Alarcon, F.C.C.P., Mexico City; Vice-President, Dr. Ismael Cosio Villegas, F.C.C.P., Mexico City; Secretary-Treasurer, Dr. Octavio Bandala, F.C.C.P., Mexico City.

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Brigadier General Shelley U. Marietta, F.C.C.P., Commanding Officer, Walter Reed General Hospital, Washington, D. C., has been promoted to the rank of Major General of the U. S. Army Medical Department. General Marietta is the Governor of the College for the United States Army Medical Corps, and he is Chairman of the Membership Committee of the American College of Chest Physicians.

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Dr. William S. Conklin, F.C.C.P., has been appointed Assistant Professor of Surgery at the University of Oregon Medical School, Portland, Oregon, and Assistant Medical Director of the University State Tuberculosis Hospital. He will also be associated with Dr. Ralph C. Matson, Portland, Oregon, in the private practice of medicine.

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Dr. George G. Ornstein, F.C.C.P., New York City, First Vice-President of the American College of Chest Physicians, has been appointed Professor of Medicine and Attending Physician (chest diseases) at the New York Polyclinic Medical School and Hospital.

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Dr. Charles P. Bailey, F.C.C.P., Philadelphia, Pennsylvania, has been appointed Clinical Assistant Professor of Surgery to the Faculty of Woman's Medical College of Pennsylvania.

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Dr. Louis H. Clerf, F.C.C.P., spoke on "Prevention of Bronchiectasis" at the annual session of the Medical Society of the State of Pennsylvania at the Bellevue-Stratford Hotel, Philadelphia, October 5-7.

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Dr. C. L. Harrell, F.C.C.P., Norfolk, spoke on "Tuberculosis in the Aged" at the meeting of the Medical Society of Virginia held in Roanoke October 25-27. "Asthmatic Atelectasis, Simulating Pneumonia" was also presented by Dr. Dean B. Cole, F.C.C.P., and Dr. L. James Buis, Richmond.

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Dr. M. H. Draper, F.C.C.P., Fort Wayne, Indiana, was elected Secretary of the Fort Wayne Academy of Medicine and Surgery at the annual meeting held May 11.

Dr. Herbert L. Mantz, F.C.C.P., Kansas City, Missouri, was elected as Councilor for the 7th District and was also appointed Chairman of the General Committee on Arrangements for the 1944 Annual Session of the Missouri State Medical Association.

Dr. G. A. Hedberg, F.C.C.P., has been appointed Superintendent of the Nopeming Sanatorium, Nopeming, Minnesota, succeeding Dr. A. T. Laird. Dr. Hedberg has been a member of the sanatorium medical staff since 1931 and in recent years has served as assistant medical director.

Dr. David S. Brachman, F.C.C.P., Detroit, Michigan, has been named one of the initial twenty-five fellows of the Aero Medical Association.

Dr. Herbert A. Burns, F.C.C.P., Minneapolis, Minnesota, has been placed in charge of the Tuberculosis Control Unit of the State Division of Public Institutions, Minnesota.

Dr. Paul M. Holmes, F.C.C.P., Toledo, Ohio, has been named President-Elect of the Medical Association of Toledo and Lucas County.

The following Fellows of the American College of Chest Physicians participated in the program of the War Time Conference of the American Hospital Association held in Buffalo on September 14: Dr. Rubin J. Erickson, F.C.C.P., Albany, presented a paper entitled "The General Hospital in Tuberculosis Control"; Dr. Nelson W. Strohm, F.C.C.P., Buffalo, discussed papers entitled "Chest Patrol" and "Tuberculous Contacts in Hospital Personnel—The Hospital's Second Line of Defense Against Tuberculosis." Dr. J. H. Donnelly, F.C.C.P., Buffalo, also discussed one of the papers presented at the program.

Dr. G. C. Bellinger, F.C.C.P., Salem, Oregon, spoke on "The Tuberclie in Relation to Clinical Medicine" at the meeting of the Central Willamette Medical Society at Eugene, Oregon, on June 3, 1943.

Major S. E. Wolpaw, F.C.C.P., Camp Breckinridge, Kentucky, was the main speaker on the program presented by the Union County Medico-Dental Society at the Sturgis Hotel, Sturgis, Kentucky, July 6, 1943. Major Wolpaw spoke on "The Diagnosis of Early Tuberculosis." X-ray films were presented to illustrate his subject.

Dr. J. B. Stocklen, F.C.C.P., East Cleveland, Ohio, spoke on "The Problem of Tuberculosis Control" at a meeting of the Ashtabula County Public Health Association.

Dr. J. A. Myers, F.C.C.P., Minneapolis, has been appointed by the Governor to serve on the medical panel provided by the last Minnesota Legislature to help decide medical questions arising in workmen's compensation cases.

## OBITUARIES

**ROBERT BROADDUS HOMAN**

1872-1943

Robert Broaddus Homan was born in Bryan, Texas, May 25, 1872. He was the son of W. K. Homan and Virginia Broaddus Homan, and was the eldest of twelve children. He received his education at Texas Christian University (formerly Add-Ran College), and was granted a degree in medicine from the Medical Branch, University of Texas, on May 15, 1897.

Dr. Homan began his practice of medicine at Colorado City, Texas, and remained there until 1905, at which time he moved to Dallas, Texas. Shortly after moving to Dallas, he developed pulmonary tuberculosis and went to Asheville, North Carolina, where he spent one year in the Winyah Sanatorium. After several months' rest in that institution his condition improved and he served Dr. Von Ruck as house doctor in the institution.

In 1907, Dr. Homan moved to El Paso, Texas, and he continued his medical work in that city until a few months prior to his death. After moving to El Paso, he limited his work to diseases of the chest and was co-owner and founder of the Homan Sanatorium, which institution he established in 1910 and continued to operate until 1936. In 1924, he erected a modern sanatorium building, which in 1937 was converted into a general hospital, now known as Southwestern General Hospital, and which is still operating under that name. Dr. Homan was co-owner and president of the Board of Directors of that institution.

At the time of his death, Dr. Homan was president of the Board of Directors of Homan & Crimen, Incorporated, operators of Southwestern General Hospital; associate medical director of St. Joseph's Sanatorium; a member of the staff of Hotel Dieu Sister's Hospital; a member of the staff of Masonic Hospital; a member of the Board of Directors of the Texas State Tuberculosis Association; a member of the Board of Appeals, Selective Service, for the El Paso District; and an elder in the First Christian Church, and formerly Chairman of its Board for a number of years. He was a member of the American Medical Association, the Texas State Medical Association, the El Paso County Medical Society, the Southwestern Medical Association, and the American College of Chest Physicians. (Charter member of Federation of American Sanatoria, the parent organization of American College of Chest Physicians.)

He was a past member of the Board of Directors of the National Tuberculosis Association; a past member of the Board of Managers of *Southwestern Medicine*, the official journal for the Southwestern Medical Association; past Associate Editor of *Diseases of the Chest*, the journal of the American College of Chest Physicians; a past member of the Board of Managers of the El Paso City-County Hospital; a charter member of the El Paso Rotary Club; a past director of the El Paso Chamber of Commerce; and past president of the El Paso County Medical Society. He was also a former councilor of the Texas State Medical Association for the first district. Dr. Homan was the author of numerous articles on tuberculosis and diseases of the chest.

Dr. Homan is survived by his wife, Mrs. Robert B. Homan, the former Miss Jennie Alexander, of Dallas, Texas, to whom he was married on January 14, 1903, and by a son, Dr. Robert B. Homan, Jr., who has been associated with his father in the practice of medicine since 1930, and by a

daughter, Mrs. Clay Gwinn, of Carlsbad, New Mexico. He is also survived by three brothers, Comdr. Ralph H. Homan, who was also associated with him in the practice of medicine, and who is now serving overseas with the U. S. Navy; Dr. C. C. Homan, of El Paso, Texas, who is a dentist; and W. V. Homan, El Paso, Texas; and by two sisters, Mrs. W. R. Smith and Mrs. Grace Beal, both of El Paso, Texas.

Chas. M. Hendricks, M.D.  
*Regent for Texas*

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### ORVILLE HARRY BROWN, M.D.

1875-1943

Dr. Orville Harry Brown was born in Kansas, July 18, 1875. He was graduated in medicine from the St. Louis University School of Medicine in 1905, and served as Assistant Professor of Pharmacology until 1907. He also received the degree of Doctor of Philosophy from the University of Chicago in 1905. From 1905 to 1907 he was associate director of the Mount St. Rose Sanatorium in St. Louis and from 1907 to 1910 he was medical director of the Missouri State Sanatorium for Incipient Tuberculosis at Mount Vernon, Missouri. From 1910 to 1916, he was Assistant Professor of Medicine at St. Louis University. In 1916 Dr. Brown moved to Phoenix where he established a private practice.

He served as Editor of "Southwestern Medicine" from 1935 to 1940. He also held the position of Historian for the Arizona Medical Society.

Despite evidences of a prostatic carcinoma discovered in 1937, Dr. Brown continued with his work and in 1939 he went, with his wife to live in Arcadia, California.

Dr. Brown wrote several articles during his illness, including a personal account of his illness entitled "The Years' Experience With Bone Cancer" which was published in "The Urologic and Cutaneous Review," June, 1942. Dr. Brown was a life member of the American College of Physicians, a diplomate of the American Board of Internal Medicine, a member of the American Association of Biological Chemists and the Royal Society of Medicine of London. He was also a charter member of the American College of Chest Physicians. Dr. Brown was the author of two books, "Laboratory Physiology" and "Asthma." He contributed numerous articles to the medical literature and he was very active in medical organizations.

Hilton J. McKeown, M.D.  
*Governor for Arizona*

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### GEORGE THOMAS PALMER

1875-1943

Dr. George Thomas Palmer was born in Springfield, Illinois, March 7, 1875. He graduated in medicine from Northwestern University and started practice in Poplar Grove, Illinois. He removed to Chicago where he engaged in the general practice of medicine. Shortly thereafter, Dr. Palmer returned to Springfield where he became interested in tuberculosis and other public health activities. He organized the Springfield Tuberculosis Association in 1911. In 1925 he built the Palmer Sanatorium which remains as a monument to his work in the tuberculosis field. Dr. Palmer was a prolific writer and many of his works have been published.

In 1917 Dr. Palmer was appointed to the position of Assistant Director of Public Health in Illinois, and he served as Chairman of the Committee on Tuberculosis in the State Council of Defense during the first World War. Dr. Palmer was a member of the Sangamon County Medical Society, the Illinois State Medical Society, the American Medical Association and was a charter member of the American College of Chest Physicians.

Dr. Palmer died at his home in Springfield on June 14 of apoplexy. He is survived by his widow, the former Miss Maude Craig of Alton, Illinois, whom he married in June, 1898, and by two brothers, Robertson Palmer, Chicago, and Brigadier General George McAuley Palmer of the U. S. Army.

Robert K. Campbell, M.D., F.C.C.P.  
Governor for Illinois

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### ROBERT MARCUS STITH

1874-1943

Dr. Robert M. Stith of Seattle died June 22 from cerebral hemorrhage, age 69 years. He was born in Galveston, Texas, in 1874. After years of study at Texas Agricultural and Mechanical College and Iowa State College, he obtained his medical degree from University of Pennsylvania School of Medicine in 1899. He interned at Presbyterian Hospital, Philadelphia, and the Marine Hospital, Savannah, Georgia. For a year he was stationed on a plantation in Hawaii. He came to Seattle in 1902, where he engaged in general practice, specializing in tuberculosis. In 1911 he became medical director of Firland Sanatorium, established and built through the generosity of H. C. Henry of Seattle, a tuberculosis institution with 200 beds. He was serving in this capacity at the time of his death. During World War I he served as captain in the Medical Corps with the 69th Artillery. He was chief of the division of tuberculosis control of the Seattle Health Department and consultant for the Marine Hospital.

John E. Nelson, M.D., F.C.C.P.  
Governor for Washington

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### JOHN COX WALL

1882-1943

Dr. John Cox Wall was born in 1882 in Macon, Georgia, and moved to Eastman with his parents when he was about 8 years of age. He was graduated from Emory University in 1907. Dr. Wall practiced medicine in Eastman and the surrounding community for 35 years. For 15 years he operated the Clinic Hospital until he became disabled a year ago. Dr. Wall served as Vice Counselor of the Medical Association of Georgia from 1922 to 1927, as Counselor from 1927 to 1935, and again as Vice Counselor until his death.

Dr. Wall was a Fellow of the American College of Chest Physicians, and the American College of Physicians.

Dr. Wall died on May 18, 1943, from heart disease. Surviving him are his widow, one son, Lt. J. C. Wall, Jr., and one daughter, Mrs. F. A. Russo, of Eastman.

James A. Redfearn, M.D., F.C.C.P.  
Governor for Georgia

**ELLIOTT I. DORN**

1877-1943

Dr. Elliott I. Dorn was born in 1877 and received his medical degree from the New York Medical College in 1904. He served on the staff of several institutions and in 1933 was licensed to practice in Newark, New Jersey, specializing in tuberculosis. For many years he was a member of the staff of the Glen Gardner Sanatorium. Dr. Dorn died on May 26, 1943, at the age of 66.

Dr. Marcus W. Newcomb, F.C.C.P.  
Governor for New Jersey

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**BOOK REVIEWS**

*Acute Infections of the Mediastinum* by Neuhof and Jemerin, The Williams & Wilkins Co., Baltimore; price \$6.00.

Feeling that a comprehensive review of this condition was long overdue, the authors present 100 cases in great detail supplemented by numerous excellent radiographs and schematic drawings. Each case is individually commented upon and the various etiologic groups are summarized and the results are presented in table form.

The authors believe that the actual frequency of these cases is not accurately reflected in the literature. They divide the cases into

- A) Those secondary to esophageal perforation
- B) Those secondary to infection of the upper respiratory passages
- C) Those secondary to infection of the lungs and pleura
- D) Those of miscellaneous etiology, such as spread upwards from the abdomen, metastatic, etc.

The authors state that the customary anatomical division of the mediastinum by artificial planes serves no useful function in an understanding of the surgical features of mediastinal infection and stress the importance of the various fascial planes which largely determine the spread of infections in this region.

They feel that immediate operation is always indicated in the traumatic group even in apparently hopeless cases, since unexpected recovery occasionally takes place. Operation is also recommended in all the non-traumatic cases which are thought to be suppurative in character, although it is recognized that considerable difficulty may frequently be encountered in making a sharp distinction between suppurative and non-suppurative cases.

The indications for cervical vs. thoracic mediastinotomy are considered and a detailed description of both types of operation is given with helpful comments upon the types of problems which are actually encountered at the operation. The analytical study shows an increasing tendency towards operative intervention in the past few years and that this has been accompanied by a considerable reduction in the mortality rate.

The authors conclude, "A mediastinal infection should no longer be regarded as a strange and rare lesion situated in a more or less inaccessible part of the body. There is no longer any justification for treating such infections in a different manner from the usual methods of treating suppurative foci. The only significant difference between the mediastinum and many other sites of infection is the absence of barriers in a

vertical direction and hence a smaller likelihood of localization of the infection. This feature obviously should be taken into account as a factor calling for earlier surgical intervention than might be imperative in some other surgical regions. Adequate drainage of the suppurative focus is the corollary. Because of the pathways of spread, however, the results of operative treatment of suppuration within the mediastinum will not be ideal, even at best, as compared with a number of infections at other sites. Hence the recognition of the stage of infection before the mediastinum is widely if at all invaded, is peculiarly the crux of the problem. The study and analysis of the cases in this series reveal clearly that the identification of that stage is usually possible. The study and analysis also reveal that the recognition of impending (or actual) invasion of the mediastinum must be based chiefly on roentgenographic study. Thus, the chief purpose of this contribution is to call attention to the necessity of recognizing situations in which invasion of the mediastinum is likely to occur to the end that appropriate surgical measures are instituted which will prevent mediastinitis. The result should be the almost complete eradication of the high mortality which characterizes mediastinitis at the present time and the shift of mediastinitis to the category of an actually rare lesion."

The presentation seems timely and well conceived and will undoubtedly help to fulfill the function which its authors hoped for it.

John W. Stacey, M.D.,

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*A Guide to Practical Nutrition.\** A series of articles on nutrition, sponsored by the Committee on Nutrition and Deficiency Diseases of the Philadelphia County Medical Society. Edited for the Committee by Michael G. Wohl, M.D., and John H. Willard, M.D. Reprinted from *Philadelphia Medicine*, 1941-1942.

Despite the enormous increase in the knowledge of nutrition since the first World War, that knowledge has not a sufficiently wide application to prevent one-third of our people from suffering the results of eating de-vitalized food. Editor Wohl asks: Should not nutrition eventually be the concern of Boards of Public Health? He states the purpose of this book thus: "*A Guide to Practical Nutrition* is to be considered part of a program of Public Health Education and not as a complete coverage of the field of nutrition. The Committee on Nutrition and Deficiency Diseases of the Philadelphia County Medical Society present the manual to the medical profession in order to increase the practicing physician's interest in the new knowledge of nutrition. They trust that in doing so they may to a degree succeed in educating the physician to the importance of educating the public to the importance of proper nutrition as a means to improved health."

This is a brief but authoritative and meaty manual, well suited to the purpose stated. It does not waste words or omit essentials. A discussion of protein starvation is particularly timely and touches upon the intravenous administration of plasma protein, amino acids and casein digests. Vitamin B Complex and Riboflavin, milled flour, diets in childhood, pregnancy, old age and in relation to dental disease—all are given more than casual mention. The appendix contains tables of food composition, weights and measures; a table of vitamin properties, functions, sources and deficiency manifestations; and hints of the conservation of vitamins

in foods. The more basic knowledge of food requirements and the metabolism of protein, fat and carbohydrates is not omitted.

The minimum of knowledge of nutrition which every practitioner of medicine should have at his finger tips, if not in mind, is accessible here in compact form. The manual deserves wide usage.

Florence A. Brown, M.D.

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\*Publication and distribution made possible through a grant-in-aid from John Wyeth & Brother, Inc., Philadelphia, Pennsylvania, as a service to the medical profession.

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*Air-Borne Infection*, by Dwight O'Hara, M.D. The Commonwealth Fund, 41 East 57th Street, New York, N. Y. 1943. 108 pages. \$1.50.

This book discusses the epidemiology of some of the more common air-borne infectious diseases, including smallpox, diphtheria, the common cold, streptococcal pharyngitis, pneumonia, and tuberculosis. These seemingly diverse conditions are grouped together because of their similar manner of distribution.

The book is in no sense a textbook. It touches lightly or not at all upon diagnosis or treatment of the condition discussed. It does not suggest or evaluate preventative measures.

Neither is the book the result or summation of any organized statistical study. Many of the conclusions in regard to changing virulence of the various organisms and individual or group resistance to disease are based upon gross mortality rate studies over the last forty years. These would appear to be very misleading, in view of the great therapeutic advances made in the same period.

By a process of specious reasoning the author arrives at some interesting but highly speculative predictions of the future course of air-borne diseases.

Waldo O. Mills, M.D.

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*Bronchiectasis*. James R. Lisa, M.D., and Milton B. Rosenblatt, M.D., New York, New York. Oxford University Press, New York. Price \$4.00.

The authors carefully studied 110 necropsy specimens in which all degrees of bronchiectasis were represented. Differential tissue stains were used extensively. They found a remarkable uniformity in the pathological findings which begin as a localized lifting up of the epithelium followed by desquamation and more or less extensive necrosis of various elements of the bronchial wall and adjacent alveoli. In the stage of repair there is a re-epithelialization of the lumen and a replacement by fibrous tissue of all the other destroyed elements.

They conclude:

- 1) That bronchial dilatation is not due to a widening of the lumen but is caused by a necrosis of the bronchial wall itself.
- 2) This produces an excavation in the lung which is in direct communication with a bronchus and is usually lined with epithelium.
- 3) Bronchiectasis is always caused by bronchopulmonary infection. The extent of the bronchiectasis depends on the virulence and distribution of the infection.
- 4) That "the presence of a destructive inflammatory reaction is suf-

ficient explanation for the ectasia without the introduction of mechanical dilating factors."

5) They discount the effect of cough, bronchial obstruction, intra-bronchial pressure and atelectasis except as these produce or contribute to a bronchopulmonary infection.

The analysis of their pathological studies and their conclusions have been expanded into a 187-page book on bronchiectasis. Chapters cover anatomy and physiology of the bronchi, pathogenesis, clinical aspects and treatment. Forty excellent illustrations, four tables and a bibliography of 220 references add to the value of the text.

The general excellence of the book should ensure for it a welcome reception.

John W. Stacey, M.D.

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*The Pleuro-Subpleural Zone.* J. Skladal, Reader in General and Experimental Pathology, Caroline University, Prague; Head of the Chest Department, Bulovka Hospital, Prague. Pp. 103, with 42 illustrations. Cambridge, at the University Press, 1942.

To an already overburdened armamentarium of physical diagnostic methods Dr. Skladal proposes still another. In this short monograph the author exhausts the subject of the corticopleural syndrome. Working on the hypothesis that chronic chest diseases are localized in the peripheral lung border at their onset, the author presents a method of auscultation which makes it possible to investigate these diseases in their latent or pre-clinical phases. The utilization of sudden expiration instead of the usual provoked expiratory cough is a point well taken and should receive favorable consideration.

By use of sudden expiration the author has been able to distinguish a distinct expiratory reduplication, tubular in character, in the presence of cortical involvement. This finding is frequently present when other means including x-ray examinations of the chest fail to detect any alteration from the normal. One chapter is devoted to experimental data supporting the clinical findings of expiratory reduplication. Chapter V is a general discussion of the corticopleural syndrome, its physiologic and pathologic basis, classification, clinical investigation by sudden expiration and x-ray and its clinical significance. There has been no attempt to discuss treatment.

Throughout the book repetition is prominent but tends to add rather than detract from the text. Whether the average clinician would be able to detect reduplication and the more rare "trebled sound" is open to question.

The book includes a summary of conclusions and an index of definitions which are excellent. Bibliography consists of a short list of references to past workers in the field. At a period when time is so important to the average physician in private practice, this book has but little to recommend it.

James T. Speros, M.D.

*The Electrocardiogram and X-Ray Configuration of the Heart.* Arthur M. Master, B.S., M.D., F.A.C.P., Cardiologist to the Mt. Sinai Hospital, New York; Assistant Professor of Clinical Medicine, Columbia University, New York. Second Edit. Pp. 404. Illustrated with 108 figures, containing 163 illustrations. Lea & Febiger, Philadelphia, 1942.

This amplified second edition fills the demand for a book primarily for the physician who takes and interprets his own electrocardiograms. Improper interpretation in tracings may be avoided if one has an appreciation of the effect upon the electrocardiogram of such factors as chest deformities, pulmonary disease, size, shape and position of the heart. Its major theses are: first, that accurate diagnosis is prerequisite to effectual therapy; second, that a technically perfect electrocardiogram must still be interpreted; third, that by juxtaposition of the teleo-roentgenogram and electrocardiogram the novice may be capable of fairly accurate interpretation; fourth, that the form of the electrocardiogram is dependent upon the size and position of the heart; and fifth, that the contour of the heart is affected by extra- and intra-cardiac factors.

The material is presented in atlas form for ease in understanding. The technical clearness of the illustrations is excellent and the clinical data are given in sufficient detail to enable correlation of the clinical and electrocardiographic factors in each case.

There is an excellent bibliography listing the important articles in each field, sources where material given in the text may be supplemented and amplified. There is in addition to an authors' index a detailed subject index.

The book is well printed, readable and understandable. It can be recommended for the general practitioner as well as the specialist in cardio-respiratory diseases.

James T. Speros, M.D.

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## ABSTRACTS

"Variation of Intrapleural Pressure with Posture in Artificial Pneumothorax" by B. G. Shapiro (Major, S.A.M.C.) Oribi Military Hospital, Natal, South Africa. *S. African Medical Journal*, March 13, 1943.

By twenty-eight observations made in sixteen patients the author demonstrates the great variation in intrapleural pressure according to the posture of the patient—whether lying prone, on back, laterally on pneumothorax side, laterally with pneumothorax uppermost, or sitting. In a second series of observations in sixteen patients he shows that extra air, varying in amount from 400 cc. to 1600 cc., was needed to bring a pneumothorax previously filled in another position up to the same pressure when the patient lay in the lateral position with the "pneumo" side uppermost. All details of pressures—respiratory variation and mean pleural—are supplied, and the conclusion drawn is perfectly clear.

He found an explanation for this in a series of x-ray pictures which show that, in the lateral position, the heart and mediastinal structures

sink under the influence of gravity to the lower side, while the diaphragm on the upper ("pneumo") side descends and assumes a more oblique position as the base of the heart moves away from the uppermost axilla. This shift does not occur in the prone, supine or sitting postures. This accounts for the fact that the intrapleural pressure in an artificial pneumothorax is minimal when the patient lies laterally with the "pneumo" side uppermost, maximal when the patient lies on the "pneumo" side, and intermediate when he is prone, supine, or sitting up.

The author suggests that refills should always be done in one position as the pressure readings obtained in different positions are not comparable. He recalls the well-known fact that unpleasant tightness in the chest after a filling can usually be eased somewhat by posturing laterally with the "pneumo" side uppermost.

This does not apply to spontaneous pneumothorax because there the distress is due mainly to the sudden shifting of the mediastinum and not to the feeling of tension in the chest.

In advocating the lateral "pneumo" uppermost position as a routine for refills he correctly cautions against a mean intrapleural pressure above zero.

David P. Marais,  
M.D., F.R.C.P., (ED.), F.C.C.P.

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"Protective Action of Eutonon on the Digitalized Heart: A Preliminary Report of Experiments on Frogs and Cats, George Zuelzer, M.D., New York, New York, *Medical Record*, Sept., 1942.

Eutonon, a solution of an extract of deproteinized liver, has been found experimentally to possess many properties and effects similar to those of digitalis. It contains no alpha-amino groups or epinephrin-like substances, is entirely free of histamine effects, and its action is quite different from that of either choline or acetylcholine. Pharmacological and clinical studies have established the hormonal character of eutonon and its physiological effects.

Like digitalis, it enhances cardiac tonus. Unlike digitalis, it increases coronary dilatation, possesses a wide margin between therapeutic dose and toxic dose, does not possess cumulative effects, does not appear to affect the central nervous system, and exerts no diuretic action.

A detailed report of experiments on digitalized frogs and cats is presented, which clearly indicates that eutonon has a protective action on the digitalized heart when the two substances are administered concurrently.

Clinically, as well, the combined use of eutonon and digitalis has been found valuable in the treatment of cardiac asthma and angina pectoris; however, in the latter condition the use of eutonon alone gave the most effective results.

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**NATIONAL COUNCIL OF TUBERCULOSIS COMMITTEES**

The National Council of Tuberculosis Committees reports that the following three states have established tuberculosis committees:

*California*

- Dr. Robert A. Peers, F.C.C.P., Chairman, Colfax  
Dr. E. W. Hayes, F.C.C.P., Monrovia  
Dr. Stephan A. Parowski, F.C.C.P., San Diego  
Dr. F. M. Pottenger, Sr., F.C.C.P., Monrovia  
Dr. Harry Warren, F.C.C.P., San Francisco

*Kentucky*

- Dr. Paul A. Turner, F.C.C.P., Chairman, Hazelwood  
Dr. Benjamin L. Brock, F.C.C.P., Waverly Hills  
Dr. Maurice G. Buckles, F.C.C.P., Louisville  
Dr. Charles J. Farrell, F.C.C.P., Covington  
Dr. John B. Floyd, Richmond  
Dr. Palmer Reed, Paducah  
Dr. Virgil Simpson, Louisville

*Maryland*

- Dr. Victor F. Cullen, F.C.C.P., Chairman, State Sanatorium  
Dr. Thomas B. Aycock, Baltimore  
Dr. Miriam E. Brailey, Baltimore  
Dr. Wm. A. Bridges, F.C.C.P., Towson  
Dr. Robert H. Riley, Baltimore  
Dr. Samuel Wolman, Baltimore

**WHITE LABORATORIES ANNOUNCE A NEW TECHNIQUE OF LOCAL CHEMOTHERAPY IN ORAL AND PHARYNGEAL INFECTIONS**

White Laboratories, Inc., Pharmaceutical Manufacturers, of Newark, N. J., currently announce an important new product, the use of which provides an improved technic for the topical application of sulfathiazole in certain oral and pharyngeal infections.

The product is White's Sulfathiazole Gum. Each pleasant-tasting tablet contains  $3\frac{3}{4}$  grs. (0.25 Gm.) of sulfathiazole.

Unique advantage of the product is the high and sustained *local* concentration of sulfathiazole its use assures. Chewed for one-half to one hour, one tablet promptly initiates and maintains a high concentration of locally active sulfathiazole throughout the chewing period, averaging 70 mg. per 100 cc. saliva. Despite this high, prolonged concentration of sulfathiazole at the site of the infection, there is but slight possibility of any systemic toxicity.

White's Sulfathiazole Gum also offers other distinct advantages over former methods of locally applying sulfa drugs in the mouth and throat. The salivary solution of sulfathiazole is maintained throughout the entire chewing period in intimate contact with infected mucosal areas which are reached only briefly, if at all, by gargles, sprays or irrigations. Moreover, none of the active ingredient is lost through induced gagging, coughing or expectoration. This new technic of oropharyngeal chemotherapy is particularly practical for the patient who insists on remaining ambulatory.

White's Sulfathiazole Gum is indicated in the local treatment of acute and chronic infections of oral and pharyngeal mucosa and contiguous tissues caused by usual "mouth varieties" of streptococcus, pneumococcus, staphylococcus and other sulfonamide-susceptible organisms; e.g., septic sore throat, acute tonsillitis, pharyngitis, infectious gingivitis and stomatitis, peritonsillar abscess, etc. Also recommended as an adjuvant to systemic chemotherapy when above conditions are complicated by or associated with pulmonary or bronchial involvement.

The usual adult dosage is one (or two) tablets chewed at intervals of one to four hours, as indicated. White's Sulfathiazole Gum is supplied in packages of 24 tablets, sanitized, in slip-sleeve prescription boxes.

*Reader Notice.*

# DISEASES OF THE CHEST

## AUTHOR INDEX

Volume IX, January-December, 1943

<i>Acosta, R. Sanchez, and D. Diaz Juan.</i> Giant Mediastinal Hernia in the Course of Bilateral Pneumothorax	166	<i>Donaldson, J. M., Jr., C. J. Koerth and R. G. McCorkle.</i> Fibrosarcoma of the Tongue with Report of a Case	151
<i>Alarcon, Donato G.</i> Extrapleural Pneumothorax—Prejudices and Facts	137	<i>Duncan, Capt. Robert E.</i> Photofluorographic Chest Survey of Navy Personnel	269
<i>Alarcon, Donato G.</i> Tuberculosis in Mexico	173	<i>Feinsilver, Oscar.</i> Change in the Rate of Circulation and Venous Pressure Following Collapse Therapy in Pulmonary Tuberculosis	514
<i>Anspach, William E.</i> Roentgenologic Aspects of Bronchiectasis	24	<i>Gemoets, Henry N., and Alvis E. Greer.</i> The Coexistence of Pathogenic Fungi in Certain Chronic Pulmonary Diseases	212
<i>Bence, Alvaro, Raul F. Vaccarezza, Alfred Lanari, Francisco Labourt and R. Gonzalez Segura.</i> The Study of the Two Lungs Separately in Practical and Research Work	95	<i>Goldman, Alfred.</i> Cavernous Hemangioma of the Lung: Secondary Polycythemia	479
<i>Bence, Alvaro E.</i> Tuberculosis in the Argentine	170	<i>Greer, Alvis E., and Henry N. Gemoets.</i> The Coexistence of Pathogenic Fungi in Certain Chronic Pulmonary Diseases	212
<i>Brennemann, Joseph.</i> Bronchiectasis as the Pediatrician Sees It	39		
<i>Browne, Col. S. M., Col. H. P. Marvin and Captain E. R. Smith.</i> Sulfadiazine Pneumonia Therapy in the Canal Zone (With Especial Reference to Bradycardia)	297	<i>Harrington, F. E., Francisco Torres, Agustin Caeiro and J. A. Myers.</i> Observations on Tuberculosis in the City of Cordoba and Pampa de Achala, Argentina, and Minneapolis, Minnesota	467
<i>Caeiro, Agustin, Francisco Torres, J. A. Myers and F. E. Harrington.</i> Observations on Tuberculosis in the City of Cordoba and Pampa de Achala, Argentina, and Minneapolis, Minnesota	467	<i>Heaton, T. G.</i> Artificial Pneumothorax in Pulmonary Tuberculosis. <i>Rev.</i>	94
<i>Carr, Duane.</i> Hazards of Pneumothorax	418	<i>Hendricks, Chas. M.</i> World Planning for Tuberculosis Control	257
<i>Clerf, Louis H.</i> Bronchoscopy in Bronchiectasis	56	<i>Hewell, Barbara A., and H. J. Nititz.</i> Woodward High School Survey	348
<i>Coyle, E. W.</i> Thoracoplasty with Apicolysis	425	<i>Hilleboe, Herman E.</i> The Tuberculosis Control Program of the U. S. Public Health Service	281
<i>Dailey, James E.</i> Intrapleural Pneumonolysis	492	<i>Hillman, Brig. Gen. Chas. C.</i> The Tuberculosis Problem in the Army	265
<i>De Bakey, Michael, and Alton Ochsner.</i> Surgical Treatment of Bronchiectasis	63		

<i>Holinger, Paul H.</i> , Symposium on Bronchiectasis, Introduction		<i>Lisa, James R., and Milton B. Rosenblatt</i> , Bronchiectasis, Rev.	540
<i>Huber, John Franklin, and Chevalier L. Jackson</i> . Correlated Applied Anatomy of the Bronchial Tree and Lungs with a System of Nomenclature	1	<i>Long, Esmond R.</i> The Relationship Between the National Research Council and the Medical Services	284
	319		
<i>Jackson, Chevalier L., and John Franklin Huber</i> . Correlated Applied Anatomy of the Bronchial Tree and Lungs with a System of Nomenclature	319	<i>Marvin, Col. H. P., Col. S. M. Browne and Captain E. R. Smith</i> . Sulfadiazine Pneumonia Therapy in the Canal Zone (With Especial Reference to Bradycardia)	297
<i>Jackson, Chevalier L.</i> Report: Council on Pan American Affairs	169	<i>Mayer, Edgar, and Israel Rapaport</i> . Contagiousness of Tuberculosis	500
<i>Jacobs, Sydney</i> . Air Embolism in Artificial Pneumothorax. Report of 3 Cases	487	<i>McCorkle, R. G., C. J. Koerth and J. M. Donaldson, Jr.</i> Fibrosarcoma of the Tongue with Report of a Case	151
<i>Juan, P. Diaz, and R. Sanchez Acosta</i> . Giant Mediastinal Hernia in the Course of Bilateral Pneumothorax	166	<i>McCullough, David</i> . Tuberculous Tracheobronchitis	448
<i>Kinghorn, Hugh M.</i> Tuberculosis of the Spine Complicating Pulmonary Tuberculosis	432	<i>Mendelsohn, Jacob J.</i> Hexylresorcinol Solution in the Treatment of Tuberculous Empyema	334
<i>Knoepp, Major Louis F.</i> Lobectomy for Pulmonary Suppuration—Revealing Foreign Body Retained for Twenty-Six Years (Case Report)	510	<i>Muller, Gulli Lindh</i> . Clinical Significance of the Blood in Tuberculosis. Rev.	381
<i>Koerth, C. J., R. G. McCorkle and J. M. Donaldson, Jr.</i> Fibrosarcoma of the Tongue with Report of a Case	151	<i>Murray, Foster</i> . Bronchogenic Carcinoma	383
<i>Labourt, Francisco, Raul F. Vaccarezza, Alvaro Bence, Alfredo Lanari and R. Gonzalez Segura</i> . The Study of the Two Lungs Separately in Practical and Research Work	95	<i>Myers, J. A., Francisco Torres, Agustin Caeiro and F. E. Harrington</i> . Observations on Tuberculosis in the City of Cordoba and Pampa de Achala, Argentina, and Minneapolis, Minnesota	467
<i>Lanari, Alfredo, Raul F. Vaccarezza, Alvaro Bence, Francisco Labourt and R. Gonzalez Segura</i> . The Study of the Two Lungs Separately in Practical and Research Work	95	<i>Navarrete, Antonio</i> . Present Tuberculosis Status in Cuba	175
<i>Lederer, Francis L.</i> Nasal Sinusitis in Relation to Bronchiectasis	50	<i>Neuhof and Jemerin</i> . Acute Infections of the Mediastinum. Rev.	538
		<i>Nimitz, H. J., and Barbara A. Hewell</i> . Woodward High School Survey	348
		<i>Ochsner, Alton, and Michael De Bakey</i> . Surgical Treatment of Bronchiectasis	63
		<i>O'Hara, Dwight</i> . Air-Borne Infection, Rev.	540

<i>Overholt, Richard H.</i> A Common Masquerading Lung Disease	197	<i>Stacey, John W.</i> The Inhalation of Nebulized Solutions of Sul- fonamides in the Treatment of Bronchiectasis	302
<i>Peabody, J. Winthrop.</i> Broncho- scopic Aids in Medical Condi- tions Within the Chest	307	<i>Taylor, Henry K.</i> The Role of the Roentgenologist in the Diag- nosis of Diseases of the Chest	115
<i>Petersen, William F.</i> Tubercu- losis: Weather and Resistance	403	<i>Terrill, Frank I., and Kenneth A. Tyler.</i> Interpleural Communica- tion in Bronchiectasis	496
<i>Rappaport, Israel, and Edgar Mayer.</i> Contagiousness of Tu- berculosis	500	<i>Torres, Francisco, Agustin Caeiro, J. A. Myers and F. E. Harring- ton.</i> Observations on Tubercu- losis in the City of Cordoba and Pampa de Achala, Argentina, and Minneapolis, Minnesota	467
<i>Riggins, H. McLeod.</i> Present Con- cepts of Pathogenesis, Morbidity, Mortality and Treatment of Bronchiectasis	5	<i>Tyler, Kenneth A., and Frank I. Terrill.</i> Interpleural Communica- tion in Bronchiectasis	496
<i>Rosenblatt, Milton B., and James R. Lisa.</i> Bronchiectasis, Rev.	540		
<i>Segura, R. Gonzalez, Raul F. Vac- carezza, Alvaro Bence, Alfredo Lanari and Francisco Labourt.</i> The Study of the Two Lungs Separately in Practical and Re- search Work	95	<i>Vaccarezza, Raul F., Alvaro Bence, Alfredo Lanari, Francisco La- bourt and R. Gonzalez Segura.</i> The Study of the Two Lungs Separately in Practical and Re- search Work	95
<i>Seligson, Frank.</i> Chemotherapy of Tuberculosis	245		
<i>Shapiro, Major B. G. (S.A.M.C.).</i> , Variation of Intrapleural Pres- sure with Posture in Artificial Pneumothorax. Abstract	542	<i>Washburn, Frank H.</i> Traumatic Pneumothorax Without Gross Injury to the Chest Wall (Case Report)	455
<i>Shaw, Robert.</i> Post-Thoracoplasty Care: Scoliosis, Pain and Re- habilitation	327	<i>Werner, Walter I.</i> A Study of the Weltermann Coagulation Reac- tion in Pulmonary Tubercu- losis	241
<i>Skladal, J.</i> , Pleuro-Subpleural Zone. Rev.	541	<i>Wolford, Roy A.</i> The Tu- berculosis Problem in the Veterans Administration	274
<i>Smith, Captain E. R., Col. S. M. Browne and Col. H. P. Marvin.</i> Sulfadiazine Pneumonia Ther- apy in the Canal Zone (With Especial Reference to Brady- cardia)	297	<i>Zuelzer, George,</i> Protective Ac- tion of Eutonon on the Digital- ized Heart. Rev.	543

## DISEASES OF THE CHEST

### SUBJECT INDEX

Volume IX, January-December, 1943

<b>A</b> IR-BORNE Infection, D. O'-Hara. <i>Rev.</i>	540	Bronchiectasis, J. R. Lisa and M. B. Rosenblatt. <i>Rev.</i>	540
Air Embolism in Artificial Pneumothorax. Report of Three Cases. S. Jacobs	487	Bronchiectasis as the Pediatrician Sees It. J. Brennemann	39
Anatomy, Correlated Applied—of the Bronchial Tree and Lungs with a System of Nomenclature. C. L. Jackson and J. F. Huber	319	Bronchiectasis, Bronchoscopy in—. L. Clerf	56
Apicolysis, Thoracoplasty With—. E. W. Coyle	425	Bronchiectasis, The Inhalation of Nebulized Solutions of Sulfonamides in the Treatment of—. J. W. Stacey	302
Argentina, Observations on Tuberculosis in the City of Cordoba and Pampa de Achala, —, and Minneapolis, Minnesota, F. Torres, A. Caeiro, J. A. Myers and F. E. Harrington	467	Bronchiectasis, Interpleural Communication in—. F. I. Terrill and K. A. Tyler	496
Argentine, Tuberculosis in the—. A. E. Bence	170	Bronchiectasis, Nasal Sinusitis in Relation to—. F. L. Lederer	50
Army, The Tuberculosis Problem in the—. Brig. Gen. C. C. Hillman	265	Bronchiectasis, Present Concepts of Pathogenesis, Morbidity, Mortality and Treatment of—. M. McL. Riggins	5
Artificial Pneumothorax, Air Embolism in—. Report of Three Cases. S. Jacobs	487	Bronchiectasis, Roentgenologic Aspects of—. W. E. Anspach	24
Artificial Pneumothorax in Pulmonary Tuberculosis. T. G. Heaton. <i>Rev.</i>	94	Bronchiectasis, Surgical Treatment of—. M. De Bakey and A. Ochsner	63
<b>B</b> ILATERAL Pneumothorax, Giant Mediastinal Hernia in the Course of—. R. S. Acosta and P. D. Juan	166	Bronchiectasis, Symposium on—. Introduction. P. H. Holinger	1
Blood, Clinical Significance of the— in Tuberculosis. G. L. Muller. <i>Rev.</i>	381	Bronchogenic Carcinoma. F. Murray	383
Bradycardia, Sulfadiazine Pneumonia Therapy in the Canal Zone (With Especial Reference to—). Col. S. M. Browne, Col. H. P. Marvin, and Capt. E. R. Smith	297	Bronchoscopic Aids in Medical Conditions Within the Chest. J. W. Peabody	307
Bronchial Tree, Correlated Applied Anatomy of the— and Lungs with a System of Nomenclature. C. L. Jackson and J. F. Huber	319	Bronchoscopy in Bronchiectasis, L. H. Clerf	56
<b>C</b> ARCINOMA, Bronchogenic—. F. Murray	383	Case-Finding in Industry. Council on Military Affairs, A.C.C.P.	288
Cavernous Hemangioma of the Lung: Secondary Polycythemia. A. Goldman	479	Change in the Rate of Circulation and Venous Pressure Following Collapse Therapy in Pulmonary Tuberculosis. O. Fein-silver	514

Chemotherapy of Tuberculosis. <i>F. Seligson</i>	245	Diseases of the Chest, "Information Please," a Quiz on—. <i>G. G. Ornstein, R. H. Overholt, O. Auerbach, and P. H. Holinger</i>	363
Chest Wall, Traumatic Pneumothorax Without Gross Injury to the—. <i>F. H. Washburn. Case Report</i>	455	<b>E</b> MBOLISM, Air— in Artificial Pneumothorax. Report of Three Cases. <i>S. Jacobs</i>	487
Circulation, Change in the Rate of— and Venous Pressure Following Collapse Therapy in Pulmonary Tuberculosis. <i>O. Feinsilver</i>	514	Empyema, Hexylresorcinol Solution in the Treatment of Tuberculous—. <i>J. J. Mendelsohn</i>	334
Clinical Significance of the Blood in Tuberculosis. <i>G. L. Muller. Rev.</i>	381	Extrapleural Pneumothorax—Prejudices and Facts. <i>D. G. Alarcon</i>	137
Coagulation Reaction, A Study of the Weltman— in Pulmonary Tuberculosis. <i>W. I. Werner</i>	241	<b>F</b> IBROSARCOMA of the Tongue with Report of a Case. <i>C. J. Koerth, R. G. McCorkle and J. M. Donaldson, Jr.</i>	151
Coexistence of Pathogenic Fungi in Certain Chronic Pulmonary Diseases: With Especial Reference to Pulmonary Tuberculosis. <i>A. E. Greer and H. N. Gemoets</i>	212	Foreign Body Retained for Twenty-six Years, Lobectomy for Pulmonary Suppuration Revealing—. <i>Maj. L. F. Knoepp. Case Report</i>	510
Collapse Therapy, Change in the Rate of Circulation and Venous Pressure Following— in Pulmonary Tuberculosis. <i>O. Feinsilver</i>	514	Fungi, Coexistence of Pathogenic—in Certain Chronic Pulmonary Diseases: With Especial Reference to Pulmonary Tuberculosis. <i>A. E. Greer and H. N. Gemoets</i>	212
Common Masquerading Lung Disease. <i>R. H. Overholt</i>	197	<b>G</b> IANT Mediastinal Hernia in the Course of Bilateral Pneumothorax. <i>R. S. Acosta and P. D. Juan</i>	166
Communication, Interpleural—in Bronchiectasis. <i>F. I. Terrill and K. A. Tyler</i>	496	Guide to Practical Nutrition. Committee on Nutrition and Deficiency Diseases, Philadelphia County Medical Society. <i>Rev.</i>	539
Compensation Claims, Contagiousness of Tuberculosis. In Relationship to—. <i>E. Mayer and I. Rappaport</i>	500	<b>H</b> AZARDS of Pneumothorax. <i>D. Carr</i>	418
Contagiousness of Tuberculosis. In Relationship to Compensation Claims. <i>E. Mayer and I. Rappaport</i>	500	Hemangioma, Cavernous— of the Lung: Secondary Polycythemia. <i>A. Goldman. Case Report</i>	479
Correlated Applied Anatomy of the Bronchial Tree and Lungs with a System of Nomenclature. <i>C. L. Jackson and J. F. Huber</i>	319	Hexylresorcinol Solution in the Treatment of Tuberculous Empyema. <i>J. J. Mendelsohn</i>	334
Cuba, Present Tuberculosis Status in—. <i>A. Navarrete</i>	175		
<b>D</b> IAGNOSIS, The Role of the Roentgenologist in the— of Diseases of the Chest. <i>H. K. Taylor</i>	115		

<b>I</b> NDUSTRIAL Army, Tuberculosis in Our—: An Appeal to Management and Labor. <i>Council on Military Affairs and Public Health, A.C.C.P.</i>	287	Mexico, Tuberculosis in—. <i>D. G. Alarcon</i>	173
Industry, Case-Finding in—. <i>Council on Military Affairs and Public Health, A.C.C.P.</i>	288	Minneapolis, Minnesota, Observations on Tuberculosis in the City of Cordoba and Pampa de Achala, Argentina and—. <i>F. Torres, A. Caeiro, J. A. Myers, and F. E. Harrington</i>	467
Infections of the Mediastinum, Acute—. <i>Neuhof and Jemerin. Rev.</i>	538	<b>N</b> ASAL Sinusitis in Relation to Bronchiectasis. A Review. <i>F. L. Lederer</i>	50
"Information Please," a Quiz on Diseases of the Chest. <i>G. G. Ornstein, R. H. Overolt, O. Auerbach and P. Holinger</i>	363	National Research Council, Relationship Between the— and the Medical Services. <i>E. R. Long</i>	284
Inhalation of Nebulized Solutions of Sulfonamides in the Treatment of Bronchiectasis. <i>J. W. Stacey</i>	302	Navy Personnel, Photofluorographic Chest Survey of—. <i>Capt. R. E. Duncan</i>	269
Interpleural Communication in Bronchiectasis. <i>F. I. Terrill and K. A. Tyler</i>	496	Nomenclature, Correlated Applied Anatomy of the Bronchial Tree and Lungs with a System of—. <i>C. L. Jackson and J. F. Huber</i>	319
Intrapleural Pneumonolysis. <i>J. E. Dailey</i>	492	<b>O</b> BITUARIES:	
<b>K</b> IRK, Norman Thomas, Major General, U. S. Army, The Surgeon General	370	Beam, Hugh A.	296
<b>L</b> OBECTOMY for Pulmonary Suppuration—Revealing Foreign Body Retained for Twenty-six Years. <i>Maj. L. F. Knoepp. Case Report</i>	510	Brown, Orville Harry	536
Lung Disease, A Common Masquerading—. <i>R. H. Overolt</i>	197	Davis, Stirley C.	295
Lungs, Correlated Applied Anatomy of the Bronchial Tree and— with a System of Nomenclature. <i>C. L. Jackson and J. F. Huber</i>	319	Dorn, Elliott I.	538
Lungs, The Study of the Two— Separately in Practical and Research Work. <i>R. F. Vaccarezza, A. Bence, A. Lanari, F. Labourt, and R. G. Segura</i>	95	Homan, Robert Broadus	535
<b>M</b> EDIASTINAL Hernia, Giant— in the Course of Bilateral Pneumothorax. <i>R. S. Acosta and P. D. Juan</i>	166	Mann, Smith J.	380
Medical Advisory Boards. <i>Edit.</i>	261	Moore, Floyd N.	380
Medical Services, Relationship Between the National Research Council and the—. <i>E. R. Long</i>	284	Palmer, George Thomas	536
		Stith, Robert Marcus	537
		Wall, John Cox	537
		Observations on Tuberculosis in the City of Cordoba and Pampa de Achala, Argentina, and Minneapolis, Minnesota. <i>F. Torres, A. Caeiro, J. A. Myers and F. E. Harrington</i>	467
		<b>P</b> AIN, Post-Thoracoplasty Care: Scoliosis,— and Rehabilitation. <i>R. Shaw</i>	327
		Peck, John H.: Biography, <i>J. A. Myers</i>	188
		Pediatrician, Bronchiectasis as the— Sees It. <i>J. Brennemann</i>	39
		Photofluorographic Chest Survey of Navy Personnel. <i>Capt. R. E. Duncan</i>	269

Pleuro-Subpleural Zone, <i>J. Skandal. Rev.</i>	541	Pulmonary Tuberculosis, Change in the Rate of Circulation and Venous Pressure Following Collapse Therapy in—. <i>O. Feinsilver</i>	514
Pneumonia, Sulfadiazine—Therapy in the Canal Zone (With Especial Reference to Bradycardia). <i>Col. S. M. Browne, Col. H. P. Marvin and Capt. E. R. Smith</i>	297	Pulmonary Tuberculosis, A Study of the Weltmann Coagulation Reaction in—. <i>W. I. Werner</i>	241
Pneumonolysis, Intrapleural—. <i>J. E. Dailey</i>	492	Pulmonary Tuberculosis, Tuberculosis of the Spine Complicating—. <i>H. M. Kinghorn</i>	432
Pneumothorax, Air Embolism in Artificial—. Report of Three Cases. <i>S. Jacobs</i>	487	<b>R</b> EHABILITATION, Post-Thoracoplasty Care. Scoliosis, Pain and—. <i>R. Shaw</i>	327
Pneumothorax, Artificial— in Pulmonary Tuberculosis. <i>T. G. Heaton. Rev.</i>	94	Relationship Between the National Research Council and the Medical Services. <i>E. R. Long</i>	284
Pneumothorax, Extrapleural— Prejudices and Facts. <i>D. G. Alarcon</i>	137	Research, The Study of the Two Lungs Separately in Practical and—Work. <i>R. F. Vaccarezza, A. Bence, A. Lanari, F. Labourt and R. G. Segura</i>	95
Pneumothorax, Giant Mediastinal Hernia in the Course of Bilateral—. <i>R. S. Acosta and P. D. Juan</i>	166	Resistance, Tuberculosis: Weather and—. <i>W. F. Petersen</i>	403
Pneumothorax, Hazards of—. <i>D. Carr</i>	418	Roentgenologic Aspects of Bronchiectasis. <i>W. E. Anspach</i>	24
Pneumothorax, Traumatic—Without Gross Injury to the Chest Wall. <i>F. H. Washburn. Case Rep.</i>	455	Role of the Roentgenologist in the Diagnosis of Diseases of the Chest. <i>H. K. Taylor</i>	115
Polycythemia, Cavernous Hemangioma of the Lung: Secondary—. <i>A. Goldman. Case Rep.</i>	479	<b>S</b> CHOOL Survey, Woodward High—. <i>B. A. Hewell and H. J. Nimitz</i>	348
Post-Thoracoplasty Care. Scoliosis, Pain and Rehabilitation. <i>R. Shaw</i>	327	Scoliosis, Post-Thoracoplasty Care. —, Pain and Rehabilitation. <i>R. Shaw</i>	327
Present Concepts of Pathogenesis, Morbidity, Mortality and Treatment of Bronchiectasis. <i>H. McL. Riggins</i>	5	Spine, Tuberculosis of the— Complicating Pulmonary Tuberculosis. <i>H. M. Kinghorn</i>	432
Present Tuberculosis Status in Cuba. <i>A. Navarrete</i>	175	Study of the Two Lungs Separately in Practical and Research Work. <i>R. F. Vaccarezza, A. Bence, A. Lanari, F. Labourt and R. G. Segura</i>	95
Public Health Service, The Tuberculosis Control Program of the U.S.—. <i>H. E. Hilleboe</i>	281	Study of the Weltman Coagulation Reaction in Pulmonary Tuberculosis. <i>W. I. Werner</i>	241
Pulmonary Suppuration, Lobectomy for—Revealing Foreign Body Retained for Twenty-six Years. <i>Maj. L. F. Knoepp. Case Rep.</i>	510	Sulfadiazine Pneumonia Therapy in the Canal Zone. (With Especial Reference to Bradycardia). <i>Col. S. M. Browne, Col. H. P. Marvin, and Capt. E. R. Smith</i>	297
Pulmonary Tuberculosis, Artificial Pneumothorax in—. <i>T. G. Heaton. Rev.</i>	94		

Sulfonamides, The Inhalation of Nebulized Solutions of— in the Treatment of Bronchiectasis. <i>J. W. Stacey</i>	302	Military Affairs and Public Health, A.C.C.P.	287
Surgical Treatment of Bronchi- ectasis. <i>M. De Bakey and A. Ochsner</i>	63	Tuberculosis in Mexico. <i>D. G. Alarcon</i>	173
Survey, Photofluorographic Chest — of Navy Personnel . <i>Capt. R. E. Duncan</i>	269	Tuberculosis, Observations on — in the City of Cordoba and Pampa de Achala, Argentina, and Minneapolis, Minnesota. <i>F. Torres, A. Caeiro, J. A. Myers and F. E. Harrington</i>	467
Survey, Woodward High School—. <i>B. A. Hewell and H. J. Nimitz</i>	348	Tuberculosis, Present— Status in Cuba. <i>A. Navarrete</i>	175
<b>T</b> HORACOPLASTY With Ap- colysis. <i>E. W. Coyle</i>	425	Tuberculosis Problem in the Ar- my. <i>Brig. Gen. C. C. Hillman</i>	265
Tongue, Fibrosarcoma of the— With Report of a Case. <i>C. J. Koerth, R. G. McCorkle and J. M. Donaldson, Jr.</i>	151	Tuberculosis Problem in the Vet- erans Administration. <i>R. A. Wolford</i>	274
Tracheobronchitis, Tuberculous—. <i>D. McCullough</i>	448	Tuberculosis: Weather and Re- sistance. <i>W. F. Petersen</i>	403
Traumatic Pneumothorax With- out Gross Injury to the Chest Wall. <i>F. H. Washburn. Case Rep.</i>	455	Tuberculous Empyema, Hexylre- sorcinol Solution in the Treat- ment of—. <i>J. J. Mendelsohn</i>	334
Tuberculosis, Artificial Pneumo- thorax in—. <i>T. G. Heaton. Rev.</i>	94	Tuberculous Tracheobronchitis. <i>D. McCullough</i>	448
Tuberculosis in the Argentine. <i>A. E. Bence</i>	170	<b>V</b> ARIATION of Intrapleural Pressure with Posture in Arti- ficial Pneumothorax, <i>Major B. G. Shapiro (S.A.M.C.) Ab- stract</i>	542
Tuberculosis of the Spine Com- plicating Pulmonary Tubercu- losis, <i>H. M. Kinghorn</i>	432	Venous Pressure, Change in the Rate of Circulation and— Fol- lowing Collapse Therapy in Pulmonary Tuberculosis. <i>O. Feinsilver</i>	514
Tuberculosis, Chemotherapy of—. <i>F. Seligson</i>	245	Veterans Administration, The Tu- berculosis Problem in the—. <i>R. A. Wolford</i>	274
Tuberculosis, Clinical Significance of the Blood in—. <i>G. L. Muller. Rev.</i>	381	Vitamins. <i>Edit.</i>	372
Tuberculosis, Contagiousness of—. In Relationship to Compensa- tion Claims. <i>E. Mayer and I. Rappaport</i>	500	<b>W</b> EATHER, Tuberculosis:— and Resistance. <i>W. F. Petersen</i>	403
Tuberculosis Control Program of the U. S. Public Health Service. <i>H. E. Hilleboe</i>	281	Weltmann Coagulation Reaction, A Study of the— in Pulmonary Tuberculosis. <i>W. I. Werner</i>	241
Tuberculosis Control, World Plan- ning for—. <i>C. M. Hendricks</i>	257	Woodward High School Survey. <i>B. H. Hewell and H. J. Nimitz</i>	348
Tuberculosis in Our Industrial Army: An Appeal to Manage- ment and Labor. <i>Council on</i>		World Planning for Tuberculosis Control. <i>C. M. Hendricks</i>	257



5

8

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